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TABLE OF CONTENTS.

I.—The Problem of Middle Ear Mechanics. By A. G. POHLMAN, M. D., from the Department of Anatomy of St. Louis University, St. Louis........ 1

II.—Intranasal Reconstruction. By F. J. PRATT, M. D., Assistant Professor, Ophthalmology and Otolaryngology, University of Minnesota Medical School, and J. A. PRATT, M. D., Instructor, Ophthalmology and Otolaryngology, University of Minnesota Medical School, Minneapolis........ 46

III.—Gravity Drainage and Swabs vs. Irrigation and Wicks in Otitis Media. By GEORGE E. BARNES, B. A., M. D., Herkimer, N. Y........107

IV.—Tonsillar Tubercles Containing Intracellular Concretions Simulating Foreign Body Pseudotubercles. By CARL VERNON WELLER, M. S., M. D., Associate Professor of Pathology, University of Michigan, Ann Arbor........110

V.—A Demonstration of the Physics of Suction Applied to the Nose with Modification of the Usual Technic. By SAMUEL IGLAUER, M. D., Cincinnati, Ohio........125

VI.—A New Local Anesthetic for Nose and Throat Work. By ALBERT E. BULSON, JR., M. D., Fort Wayne, Indiana....131

VII.—A Study of the Schwabach Test in One Hundred Cases. By ROBERT SONNENSCHEIN, M. D., Chicago........137

VIII.—Tonsillectomies in Adults for Rheumatism with Critical Review of Results. By HILL HASTINGS, M. D., Los Angeles, California........157

IX.—Hyoscin and Morphin as a Preliminary to Local Anesthetics. By LEE M. HURD, M. D., New York........167

X.—Nasociliary Neuralgia. By GREENFIELD SLUDER, M. D., St. Louis........172

XI.—Vocational, Occupational or Industrial Diseases of the Ear, Nose and Throat. By JOHN A. DONOVAN, M. D., Butte, Montana........176

XII.—Tinnitus Aurium. By THOMAS J. HARRIS, M. D., New York........192

XIII.—Postoperative Treatment of the Radical Mastoid Operation. By J. A. Stucky, M. D., Lexington, Ky..........200

XIV.—Tic Douloureux in Relation to Latent Maxillitis. By H. B. Lemere, M. D., Omaha, Neb..........204

XV.—Dichloramin Treatment of Mastoid Wounds. By Ralph A. Fenton, A. B., M. D., Portland, Ore..........214

XVI.—Acute Lacunar Adenoiditis. By Samuel Salinger, M. D., Chicago..........220

XVII.—The Problem of the Hard of Hearing and Electrical Hearing Devices. By George E. Shambaugh, M. D., Chicago..........225

XVIII.—The Bacteriology and Histopathology of the Adenoids. From the Department of Pathology and Bacteriology of the University of Illinois, College of Medicine, Chicago. By Isadore Pilot, M. D., Chicago..........231

XIX.—Fatalities Following Operations Upon the Nose and Throat Not Dependent Upon Anesthesia—A Study of Three Hundred and Thirty-two Hitherto Unreported Cases. By Hanau W. Loeb, M. D., St. Louis..........273

XX.—Optic Nerve and Accessory Sinuses. By Prof. J. van der Hoeve, Leiden, Holland..........297

XXI.—Recent Observations on Laryngopulmonary Tuberculosis. By Julius Dworetzky, M. D., Liberty, N. Y..........311

XXII.—The Use of Ear Plugs in Aviation. By Major Lloyd E. Tefft, Medical Corps, U. S. A., and Elizabeth K. Stark, Department of Otology, Medical Research Laboratory and School for Flight Surgeons, Mitchell Field, L. I., N. Y..........329

XXIII.—Larvae of the Fly Sarcophaga (Sarcophila) Wohlfahrtii in the Ear. By Alexander Zebrowski, M. D., New York..........335

XXIV.—Adenocarcinoma of the Middle Turbinate, Antrum and Ethmoids Two Years After Radical Operation Without Recurrence. By David N. Husik, M. D., Philadelphia..........345

XXV.—Chronic Stenosis of Larynx and Trachea With Report of a Case Treated by Laryngostomy and Dilatation. By Edward Cecil Sewall, A. B., M. D., San Francisco..........352

XXVI.—The Histopathology and Histogenesis of Benign Growths of the Nose and Accessory Cavities. By HARRY L. BAUM, M. D., Denver 371

XXVII.—Nasal Tuberculosis. By W. B. CHAMBERLIN, M. D., Cleveland 423

XXVIII.—The Problem of Middle Ear Mechanics. Chapter II. By A. G. POHLMAN, M. D., From the Department of Anatomy of St. Louis University, St. Louis 430

XXIX.—Misconceptions Regarding the Import of Retraction of the Drum Membrane. By EDMUND PRINCE FOWLER, M. D., New York City 483

XXX.—Acute Infections into the Sebaceous Glands and Hair Follicles of the Nasal Vestibule. By LEE M. HURD, M. D., New York 489

XXXI.—Variations of Intracranial Symptoms From Frontal Sinusitis. By LOUIS LEVY, M. D., F. A. C. S., Memphis, Tennessee 493

XXXII.—Acute Mastoiditis With Unusual Complications. By E. P. NORCROSS, M. D., Chicago 501

XXXIII.—The Role of the Tonsils in the Treatment of Nose Conditions, Infective and Obstructive—Study of 111 Cases. By T. R. GITTINS, M. D., Sioux City, Iowa 507

XXXIV.—Suppurations of the Labyrinth. By EDWIN COBB, M. D., Marshalltown, Iowa 523

XXXV.—The Application of Dental Molding Compound for the Maintenance of Skin Grafts in the Middle Ear and Mastoid Cavities. By JOSEPH ISRAEL, M. D., Houston, Texas 543

XXXVI.—Some Remarks on the Indications for Simple Mastoid Operation in the Adult. By JOHN B. RAE, M. B., C. M., New York 546

XXXVII.—Specimens Illustrating Operative Aural Surgery and the Making by Graduate Students of Preparations Showing Aural Surgical Anatomy. By E. B. GLEASON, M. D., Philadelphia 552

XXXVIII.—The Treatment and Prognosis of Carcinoma of the Larynx. By WAICHIRO OKADA, M. D., Professor of Oto-Rhino-Laryngology in the Imperial University of Japan, Tokyo, Japan 601

XXXIX.—The Study of the Tonal Ranges in Lesions of the Middle Ear. By L. W. DEAN, M. D., and C. C. BUNCH, Ph. D., Iowa City, Iowa.....617

XL.—Observations on Hay Fever. By I. CHANDLER WALKER, M. D., Boston.....600

XLI.—Unpleasant End-Results in Well Performed Tonsillectomies. By CHARLES W. RICHARDSON, M. D., Sc. D., Washington.....678

XLII.—A Contribution to the Study of Brain Abscess. By J. K. MILNE DICKIE, M. D., Ch. B., F. R. C. S. Edin., Ottawa, Canada.....683

XLIII.—The Intimate Lymphatics of the Trachea. By JEROME F. STRAUSS, M. D., Chicago.....715

XLIV.—Malignant Tumor of the Temporal Bone. By ALEXANDER ZEBROWSKI, M. D., New York.....739

XLV.—A Brief Review of the Anatomy and Surgery of the Temporal Bone, with Reference to the Mastoid in Health and Disease. By J. C. KEELER, M. D., Philadelphia.....759

XLVI.—Sphenoethmoid Sinusitis. By JOHN J. SHEA, M. A., M. D., Memphis.....813

XLVII.—The Present Status of Skiagraphic Interpretation as an Adjunct in the Diagnosis of Catarrhal Affections of the Accessory Sinuses. By ROSS HALL SKILLERN, M. D., Philadelphia.....855

XLVIII.—Otitic Pyemia Without Sinus Thrombosis. By ERNEST M. SEYDELL, M. D., Wichita.....862

XLIX.—Report of a Case of Thrombosis of the Superior Petrosal Sinus of Otitic Origin, Presenting a Symptom Complex Strongly Suggesting the Diagnosis. By W. G. SHEMELEY, JR., M. D., Philadelphia.....869

L.—Electrocoagulation in the Treatment of Carcinoma of the Larynx. By FRANK J. NOVAK, M. D., Chicago.....878

LI.—Vasomotor Disturbances of the Upper Air Passages and Sinus Disease. By J. L. GOODALE, M. D., Boston.....882

LII.—Barany's Priority. By IRVING WILSON VOORHEES, M. D., New York City.....895

LIII.—Mishaps in the Puncture and Irrigation of the Maxillary Sinus. By W. E. GROVE, M. D., Milwaukee.................913

LIV.—Surgery of the Ethmoid Labyrinth. By ALBERT H. ANDREWS, M. D., Chicago.................947

LV.—The Pathologicoanatomic Difference Between the Fetid and the Nonfetid Ozena. By ALFRED RUNDSTROM, M. D., Chicago.................950

LVI.—Abscess of the Lung and the Method of Prevention in Nasopharyngeal Surgery. By CHARLES W. RICHARDSON, M. D., Sc. D., Washington.................960

LVII.—Hyperplasia and Infection in Postethmoidsphenoid Ocular Complications. By G. F. HARKNESS, M. S., M .D., Davenport, Iowa.................964

LVIII.—Some Phases of Esophageal Stenosis. By RICHMOND MCKINNEY, M. D., Professor of Otolaryngology, University of Tennessee College of Medicine, Memphis.................977

LIX.—Interesting Brain Abscess of Rather Long Duration. By C. J. ADAMS, M. D., Kokomo, Ind.................984

LX.—A Septic Type of Temperature Not Referable to the Ear in Cases of Acute Suppurative Otitis Media. By HAROLD I. LILLIE, M. D., Section on Otolaryngology and Rhinology, Mayo Clinic, Rochester, Minn.................990

LXI.—Extradural Abscess Complicating Frontal Sinusitis—Report of a Case. By ROSS HALL SKILLERN, M. D., Philadelphia.................997

LXII.—A Method of Demonstrating the Surgical Anatomy of the Mastoid by Models. By JESSE WRIGHT DOWNEY, JR., M. D., Baltimore.................1009

LXIII.—The Technic of Thyrotomy. By ARTHUR E. HERTZLER, M. D., Halstead, Kansas.................1013

LXIV.—Subcutaneous Emphysema of the Neck and Chest Following Tonsillectomy in an Epileptic. Recovery. By SYLVAN ROSENHEIM, M. D., from the Nose and Throat Department of the Hebrew Hospital, Baltimore.................1027

LXV.—A Case Showing Unusually Early Metastasis in Carcinoma of the Vocal Cords. By ARTHUR E. HERTZLER, M. D., Halstead, Kansas.................1032

LXVI.—A Fatal Case of Vincent's Angina. By DAVID N. HUSIK, M. D., Philadelphia.....1039

LXVII.—X-Ray Treatment of Tonsillar and Lymphoid Tissue. By MAJOR JOHN H. TRINDER, M. C., U. S. Army Station Hospital, Fort Sam Houston, Texas.....1044

LXVIII.—Observations Upon the Formation and Function of the Accessory Nasal Sinuses and the Mastoid Cells. By ARTHUR W. PROETZ, M. D., Instructor in Clinical Rhinology and Laryngology and Assistant in Clinical Otology, Washington University School of Medicine, St. Louis.....1083

LXIX.—Operation of Total Laryngectomy for the Cure of Intrinsic Cancer of the Larynx. By JOHN E. MCKENTY, M. D., New York.....1101

LXX.—Abscesses of the Larynx and Trachea Following Influenza. By CLEMENT F. THEISEN, M. D., Albany.....1118

LXXI.—Some Points in the Comparative Anatomy of the Nose and the Accessory Sinuses Which Account for the Variations in These Structures in Man. (Lantern Slides.) By J. M. INGERSOLL, A. M., M. D., Professor of Otolaryngology in the Medical Department of Western Reserve University, Cleveland.....1123

LXXII.—Intranasal Injection of Alcohol in the Treatment of Hyperesthetic Rhinitis and Some of the Nasal Neuroses. By OTTO J. STEIN, M. D., Chicago.....1129

LXXIII.—The G. E. Barnes Drum Membrane Knife and Spatula. By GEORGE E. BARNES, B. A., M. D., Herkimer, N. Y.....1137

SOCIETY PROCEEDINGS.....239, 558, 899, 1139

Chicago Laryngological and Otological Society.....239, 252, 558, 573, 899, 1139, 1154

Joint Meeting of the Chicago Medical and Chicago Laryngological and Otological Societies.....264

Nashville Academy of Ophthalmology and Otolaryngology.....593, 1163

Abstract of the Scientific Proceedings of the Forty-fourth Annual Congress of the American Laryngological Association.....1166

ANNALS
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INCORPORATING THE INDEX OF OTOLARYNGOLOGY.

VOL. XXXI. MARCH, 1922. No. 1

I.

THE PROBLEM OF MIDDLE EAR MECHANICS.

BY A. G. POHLMAN, M. D.,

FROM THE DEPARTMENT OF ANATOMY OF ST. LOUIS UNIVERSITY,
ST. LOUIS.

The paper has been divided into three chapters. This has been done to make the article less cumbersome and to do away with any idea of a monographic treatise. The bibliography has been reduced to a minimum of the more important articles cited.

The subject matter of the first chapter includes the following topics:

1. The interpretation of relation of structure and function and the importance of comparative study.
2. A brief statement of the structural complex of the middle ear in anurans, birds and mammals.
3. A description of the passive traction system of elastic ligaments and their relation to middle ear musculature and displacements of the sound transmission system.
4. A statement of the theory-group, which may be termed the theory of the indirect activation of the end organ illustrated by the Wrightson theory.

5. The proposal of seven leading questions which together with the answers constitute serious objections to the theory of indirect activation, in particular to the Wrightson theory.

Chapter II of this article will take up:

1. A summary of the seven questions to make Chapter I overlap Chapter II.
2. A statement of wherein the theory of direct activation opposes all theories of indirect activation.
3. The mechanics of the diaphragm-rod theory of sound transmission.
4. The relation of a complicated apparatus to the damping effect on sound transmission.
5. An interpretation of middle ear mechanics of anurans, birds and mammals dependent on a middle ear function apart from that of sound transmission.

Chapter III will consist in:

1. A summary of the subject matter in Chapter II in the form of answers to the questions asked in this chapter.
2. Interpretation of air and bone transmission of sound.
3. A consideration of wherein the diaphragm-rod theory of direct activation lends itself to an explanation of—
    a. The Weber phenomenon.
    b. The Schwabach test.
    c. The Gelle observation.
    d. The Rinne-negative group.
    e. The instances of excellent hearing with loss of drum membrane and ossicles.
    f. The condition of paracousis Willisi.
    g. The loss of hearing in otosclerosis.

The writer wishes to extend his thanks for helpful suggestions and assistance to many colleagues, in particular to Father de Laak, Father Gruender and Mr. Carl Miller of St. Louis University; to Prof. A. L. Foley and Prof. W. J. Moenkhaus of Indiana University and to Dr. F. B. Jewett of the Research Department of the Western Electric Co. of New York City.

CHAPTER I.

It is the writer's purpose to bring the general aspects of this very complicated problem into a reasonably accessible and readable form. The physical, anatomic and physiologic material presented is designed to place the reader in a position to judge for himself whether or not the present accepted explanations fit the facts. The problem itself merits and demands the attention of clinical observers.

The terms "structural adaptation" and "functional adaptation" are rather loosely employed to cover certain deficiencies and errors in observation and interpretation. When a given region displays an anatomy which accounts satisfactorily for the functional results, we are wont to speak of it as "structurally adapted." When the physiologic results are not distinctly correlated with the structural complex, we regard the organ as "physiologically adapted." It may be that these terms are often regarded as interpretations of facts when they represent for the most part a conception of the limitations of knowledge. Structure and function, like the chick and the egg, arise synchronously, and are difficult to separate in one celled forms. A structure does not appear because of a physiologic demand nor is a result in function necessarily associated with the anatomic character of the organ.

The correlation of structure and function is one of the most difficult problems in natural science and involves asking the question "Why?" In answering this question both anatomist and physiologist are driven back to a catch-all system of terms comprising a group of phenomena spoken of as "vital manifestations." In other words, the answer is: "We don't know." However, in approaching the boundary line of "vital manifestations," the terms "structural adaptation" and "functional adaptation" are coined, giving the impression that they really explain something. As a matter of fact, these terms are admissions that cause and effect closely resemble each other. The two terms may be used almost interchangeably in an explanation. Structural factors are naturally more readily associated with adaptive results than are functional requirements accounted for by structural changes. In other words, it is more logical to assume that the camel wandered out on the desert to escape its enemies because of its peculiarities in structure than to argue it wandered out on the desert to escape its enemies and became structurally modified. The desert adaptation is therefore a cause or a result? It is more likely a cause, although considering it as a result appears to offer an "explanation" which is satisfactory to the majority of people.

The translation of a structure which results in a modified function or the apparent development of a new organ to anticipate a function is one of the most baffling of all developmental problems. Yet this much we know in a reasonably definite way. The entire vertebrate anatomy is built up on a more or less fixed plan. Every form of animal shows at least two types of organs; first, an active functional system of organs which may be definitely correlated as useful and connected with the survival of the form; and second, a less active, or perhaps nonfunctional system of organs which are not definitely useful and perhaps not definitely correlated with survival. This latter class of organs is made up of at least two varieties of structures. First, organs which are rudimentary in character but which at one time functioned as organs of the first variety. These are of historic interest and their former function is more or less easily determined because of the structural history (phylogeny). The second variety of nonuseful organs comprises a limited class which are perhaps "forerunners," not dependent on past or present requirements. These are undoubtedly related to the possibilities of the future in structural variations upon which "sport forms" may be derived. This last named class of structures would be indeed difficult to list in the human being because we have no knowledge of the possibilities in his future modifications.

If we disregard structural variations of doubful future importance it follows that the present function of any system of organs is in part dependent on its ancestral requirements. Just so the structure of any system is related to the anatomic pattern upon which the vertebrate is constructed. For example, the olfactory apparatus is in no direct way associated with the function of the respiratory tract. It preserves its ancestral relation to the digestive tract and the genital system as first found in the fishes. With the structural modification of an aquatic form into a terrestrial vertebrate, the olfactory area became folded into the new respiratory passage concomitant with lung respiration. It is "functionally adapted" to preserve its old manner of receiving and transmitting impulses, and the requirement of a watery medium is maintained in the development of a new glandular apparatus. This preservation of a definite type of ancestral organ is also paralleled in the survival of ancestral functions for which there is neither a direct anatomic nor a physiologic explanation. The "ear-cough reflex" elicited by tickling the drum membrane or brought about through middle ear disease, may not be explained except as a survival of a respiratory reflex mechanism present in the fishes and amphibians and perhaps of no great physiologic value in man. A persistence of phylogenetic structure and phylogenetic function must therefore be carefully borne in mind. Both structure and function may be found in a system without reference to its apparent office at the present time. The older requirements may be entirely overlooked because the "adaptations" appear to be complete and the present relation of structure to function too plainly obvious. Facts are one thing and the interpretation of facts is dependent on the personal equation and limitations in the observer. The reader will please bear this last sentence in mind in considering the subject matter to be presented and the deductions which are to follow.

A consideration of middle ear mechanics has usually been directly associated with the function of the inner ear. The middle ear region itself has, however, a complicated anatomic and physiologic hsitory. This history must be included in the determination of its function apart from the significance of a relation to acuity in hearing or to the mechanics of the inner ear itself. It is obvious therefore that the comparative anatomy and physiology must be carefully considered before we may separate that which is of phylogenetic importance from that which has a definite relation to newer structures and functions. In other words, a correlation of the comparative anatomy and physiology must be established before we can properly evaluate the mechanics of the middle ear itself as opposed to that associated with the inner ear and end organ.

It will be recalled that the middle ear, tuba and drum membrane arise in terrestrial anurans. The relation of structure to function is at the present time under investigation by a number of writers. The mechanics of the frog's ear are apparently complicated by a series of factors:

First. By the respiratory function of the middle ear region as determined by its blood supply through the pulmocutaneous vessels tributary to the left auricle. It is also demonstrated by the observation of actual displacements of the surface drum membrane which occur during respiration.

Second. In the development of a fenestra cochleæ (round window) which is not in relation to the tympanic cavity but is located at the base of the skull and applied to a lymph sinus.

Third. In the wide dehiscence which exists in the otic capsule on its subdural aspect.

Fourth. In a peculiar regulator muscle which arises from the suprascapula and attaches to a skeletal element (operculum) functionally interposed between the medial end of the sound transmitting apparatus (columella) and the perilymph of the vestibule.

And finally, in a wide open tuba auditiva which connects the cavum tympani to the mouth cavity. It appears as soon as the metamorphosis of an aquatic form into one of the terrestrial habit occurs (tadpole into frog) certain changes take place in the inner ear apparatus, namely, the development of a second analyzing area for the appreciation of sound vibration. This represents a new functional requirement concomitant with new physical requirements because the watery medium is not physically constituted for the natural origin of sound itself.

The old type of inner ear, with the progressive development of semicircular canals and its undoubted reception of noises and pressure appreciation found in the fishes, is developed in the direction of sound appreciation in the tadpole before it leaves the water. The region of the first branchial pocket is modified into a tuba, middle ear and drum membrane. Further, a cartilaginous rod is interposed between the drum membrane and inner ear to furnish a transmission system for sound vibrations to the perilymph (fenestra vestibuli).

Apparently another requirement is met in the origin of the second window (fenestra cochleæ). This window does not develop in relation to the tympanic cavity at all, but seems to allow for a mass displacement in the perilymph due to the apposition of movable skeletal elements (operculum and columella) to the liquid of the inner ear. The operculum is functionally interposed between the columella and the perilymph and has a muscle attached to it which arises from the suprascapula. The operculum is provided with a stout elastic ligament which undergoes stretching during the muscle pull. The ligament returns the skeletal element to a position of rest on relaxation of this muscle. The drum membrane displacements brought about through active injection of air into the tympanic cavity during breathing are geared down in the columellar system. For this reason, movements at the footplate are markedly reduced in proportion to drum membrane displacement. The action of the opercular muscle appears to inhibit marked inward thrusts of the columellar system. It therefore probably takes care of excess pressure propagation from the drum membrane or prevents an excessive mass displacement in the perilymph proper. It is interesting to note that in higher forms with later functional tubal closure, the wide dehiscence in the otic capsule adjacent to the subdural space becomes pronouncedly reduced—practically to a aqueductus vestibuli. The round window in higher forms, with certain exceptions, rotates to face the tympanic cavity. In any event, the perilymph pressure in the amphibians is directly related to intracranial pressure and to blood pressure. Displacement factors in the perilymph are therefore necessarily spread over a relatively enormous area.

It is necessary to eliminate the reptile family from this brief phylogenetic survey because the forms, such as snake, lizard and turtle, are extremely variable, and because our information on their middle ear mechanism is not well understood. The reactions of the inner ear to the mechanic factors in sound transmission, particularly in comparing the aquatic turtle forms with terrestrial varieties, are extremely interesting but may not be taken up in this paper.

Birds, on the contrary, show great uniformity in the structure of the middle ear apparatus and represent a form which the writer has investigated somewhat extensively. The conclusions on the middle ear mechanics may be stated in very general terms after certain points of similarity and dissimilarity to those of the mammal have been discussed.

While the tympanic cavity and transmission system are very different in the bird to that in the mammal, there can be little question regarding the acuity in hearing. The one prominent feature in the bird's middle ear which points to a similar function to that in mammals is this: The gross drum membrane displacements do not give rise to correspondingly marked movements at the perilymphatic end of the sound transmitting apparatus—at the fenestra vestibuli. It is a matter of fundamental importance that some comparison be made of this region in birds and mammals, because of a marked similarity in function and an equally pronounced dissimilarity in structure. The bird, like the mammal, has a functional separation of the middle ear region from the mouth or nasopharynx in a tuba auditiva which is usually closed. The general differences may be discussed under several headings:

First: The tubæ of birds open by a small, short, common duct into a recess in the roof of the mouth which is bordered by palatal folds and muscles. The animal may be said to have a sort of functional cleft palate which appears to shear off the tubal recess and respiratory area of the nose during swallowing. This may in part account for the curious manner of drinking. The stout walled common tubal duct occupies a foramen between the sphenoid and occipital bones. It immediately forks into two bony tubæ auditivæ which lead into the right or left tympanic cavities. A movable cartilage tube or the direct influence on the tubal wall, as described in the raising of the soft palate in the mammal, does not occur. The tubal duct, notably in the goose, is provided with a stout involuntary musculature which appears to function in part as a sphincter. Definite elastic ligaments, which again in the goose are largely smooth muscle, are prolonged up the tubæ from the region of the tubal duct. The function of this apparently skeletal involuntary muscle is not known at the present time. The opening of the tubal duct is therefore not comparable to the opening of the tuba in the mammal. It may be said, however, that the recess in the mouth bears a somewhat suggestive comparison to the described function of a soft palate.

Second: The pneumatization of the bird's skull through the tubæ and middle ear is especially extensive. This results in an obvious lightening of the skull, which might immediately suggest itself as a structural adaptation. The causal factor in the air sinus development is probably quite a remote one and must be mentioned if but briefly. The origin of the air sinuses is probably due to the same sort of factor which occasions the pneumatization of other bones in the bird associated with respiratory function and with the curious manner of rebreathing the air through the lungs. It may be well to consider this point, because the bird's accessory sinus system lends itself well to a comparison with the respiratory middle ear in frogs. The air sinus system may therefore represent, more or less, an earmark of recapitulation in the mammal, appearing late and of feeble respiratory nature. It is not proper to dismiss the cause of air sinuses with the lightening of the skull result on the basis that a poor explanation is better than none. It is the writer's opinion that the persistence of an ethmoidal labyrinth is of a distinct mechanical disadvantage in the primate. It appears that with ventral rotation of the eyes came a medial displacement of the orbital walls until finally the two medial walls became parallel or roughly so. However, the marked divergence of the orbital axis does not allow for the coordinated eye movement in the primate, in whom parallelism arises because of a fovea and a fusion center. The result is that parallelism is only attained by muscular action, and accounts for the lateral shifting of each eye during sleep, after death, or even in the far away look of inattention.[32]

If we regard the air sinuses as phylogenetic remnants, the enormous variation in development and the readiness of their infection from the pathologic nose of the modern civilized state at once becomes more easily understood. Not only is the air sinus system connected with the tuba and middle ear of birds much better developed, but the right and left systems are confluent across the middle line through absorption of bony partitions. These partitions are absorbed before the hatching of the chick—i. e., before the spaces are ever exposed to air. The responsibility for the origin of these spaces must therefore undoubtedly be due to at least two factors: one of ancestral character, because of the structural pattern; and the other, as an adjustment to the replacement of excess bone development dependent on the thrusts and stresses imposed through weight and muscular traction. Apart from the mastoid region, which displays marked reaction to the pull of the M. sternomastoideus, the air sinus region of the mammal is curiously devoid of stout muscular attachments. There is a suggestion that air sinuses are a result, not a cause of the bone absorption.

The enormous surface area of the large confluent air sinus system, when associated with the minute single tube-duct orifice in birds, permits the establishment of a mechanical factor connected with the drum membrane region in two ways:

First. The probability of air absorption from this large surface creating a negative pressure on the drum membrane.

Second. The great amount of air contained and the minute tubal orifice expose the bird's middle ear region, particularly the drum membrane, to large barometric fluctuations. The very fact that the amount of air contained and the amount of surface do not bear a definite relation to the size of the drum membrane makes the problem an interesting one. This is particularly true in comparing the mechanics of this region in birds and mammals. It will at least be admitted that the bird's drum membrane is exposed both through habit and structure to greater possibilities in barometric fluctuations than in the mammal. This point will be elaborated in greater detail later when certain other structural features have been discussed and their correlation with the bird's drum membrane mechanics considered.

Third. The drum membrane in birds is relatively larger than in mammals and more delicate in construction, particularly in its anterior portion. It is also more movable as a whole than that of the mammal. This mobility in the drum membrane, however, does not arise on account of its attachment. While an annulus tympanicus is wanting, the surrounding bony area is thoroughly fixed except in front, where the drum membrane abuts on the quadratum. This quadrate bone in the birds, for the benefit of those whose memory of comparative anatomy may be somewhat hazy, is intermediate between the mandible and the head. It also participates in a swinging motion, with movements of the upper bill, particularly in some birds like the parrot. This bone is either suppressed in passing from bird to mammal or is transformed, together with the articulare, into the malleus-incus, which may account for the curious relation of the processus anterius mallei to the fissura petrotympanica. The definite arrangements of the circular and radial drum membrane fibers, regarded as dampers or as inhibitors to periodic drum membrane vibrations by the Helmholtz school, are not found definitely in birds. Neither is the umbo of the mammal, regarded as a sort of inverted megaphone, and held as extremely important by some in the catchment of sound vibrations, present. The bird's drum membrane is convexed outward—roughly at right angles to the axis of the external auditory canal. The drum membrane in birds is capable of much greater inward and outward excursion than is that of the mammal with its very restricted movements in this respect. On negative pressure, the drum may come to apply its anterior surface to the wall of the external canal. On positive pressure, the same segment may flatten against the medial wall of the tympanic cavity. It will appear from this alone that the fixity, tension, structure and position of the mammalian drum membrane in terms of sound catchment efficiency may be strongly questioned when compared to the indisputable evidences, at least end result evidences, of the highly efficient membrane in birds. It may be possible that too much stress is placed on a plausible interpretation rather than on one which will harmonize with the fundamental facts and relations.

Fourth: Unlike the mammal, the bird possesses but one voluntary muscle attached to drum membrane and sound transmitting apparatus. This muscle was first described as a laxator of the drum, but later investigators seemed to prove it to be a tensor. It is accordingly named the M. tensor tympani, which perhaps is unfortunate, as will be developed later. The muscle arises outside the skull near the occipital condyle. It passes through a foramen into the territory of the external auditory canal to insert by tendinous fibers into the dorsal inferior drum quadrant and the lateral cartilage apparatus of the sound transmitting system (extracolumella). The muscle function has been carefully examined by Breuer,[7] who describes the action as a drum tensor and also one which displaces the columellar apparatus out of the oval window and thereby decreases intralabyrinthine pressure. He calls attention to an important point: that if this single muscle functions in a manner which replaces the M. tensor tympani and M. stapedius in the mammal, then the two latter muscles cannot be regarded as opponents but as synergists. The writer has called attention to certain unconsidered factors in relation to this peculiar muscle, and the reader is referred to another article[33] for the details of description or function of the elastic ligament system associated with the drum membrane and sound transmitting apparatus in birds.

Our conclusions on the bird's middle ear were that the muscle does oppose a positive air pressure against the drum membrane. It supports not only the drum membrane but also the columellar system from pronounced medial displacement. The negative pressure against the lateral surface of the drum membrane is opposed in a similar way through the elastic ligament system, which will be discussed later under the passive traction system related to the drum membrane and columellar apparatus.

The M. tensor tympani and M. stapedius arise in the mammal concomitant with a three ossicle system replacing a columellar apparatus. Phylogenesis would indicate that the M. stapedius is analogous, if not homologous, with the M. tensor tympani of birds and the M. opercularis of the amphibians. The M. tensor tympani and its associated M. tensor veli palatini (dilator tubæ) arise as new structures derived from the mastication muscle complex as shown by Eschweiler.[12] They are therefore innervated direct by the N. trigeminus—not otic ganglion, as usually described. The functional interrelation of the M. tensor tympani and M. dilator tubæ has been hinted at by a number of writers, but has not been satisfactorily established.

The function of the M. tensor tympani and M. stapedius is variously described, and demands at least a few words, because it is important to the problem of middle ear mechanics. We offer as a suggestion that it may be possible to combine all of the described functions of these two muscles without seriously offending one or another school and perhaps clarifying an obscure physiologic point.

Politzer[34] first described these two muscles as opponents or antagonists. The M. tensor tympani through its contraction increases the drum membrane tension by displacing the malleus and incus and therefore the stapes toward the perilymphatic space. The active opposition to this medial displacement is offered by the M. stapedius in rocking the ventral end of the stapes away from the vestibule. There are certain anatomic and physiologic objections to this theory of the antagonism of M. tensor tympani and M. stapedius.

The second viewpoint is admirably shown by Kato[21] in an exhaustive study of reactions, mainly in cat and rabbit. Kato found that both of these muscles may react to auditory stimuli at the same time. He regards them as synergists. He agrees therefore with Breuer's contention for birds, except that he does not regard the tension of the drum membrane as anything other than an incidental effect. Kato holds the muscles are both set in to afford protection against prolonged auditory irritation. He could not demonstrate in many trials in rabbits and cats a single visible displacement of the drum membrane due to contraction of the M. stapedius. The reciprocal innervation, physiologically essential to antagonistic muscles, does not appear to be confirmed on the basis of Kato's excellent researches. Nor is antagonistic action in keeping with the condition found in birds where but a single muscle is present.

The third point of view might be stated as independence of contraction of the M. tensor tympani and M. stapedius, and on this only incomplete data are at hand. It appears, however, that with a single muscle and elastic ligaments in a bird, replaced by an ossicular chain and two muscles in the mammal, another possibility arises. If the M. tensor tympani draws the drum membrane in, then there must be some force which pushes the drum membrane out—not the contraction of the M. stapedius. If the M. stapedius displaces the footplate outward there must be some force against which it operates which displaces it in again (probably not entirely the contraction of the M. tensor tympani). If we grant a positive thrust factor is present, then Mangold's[27] interesting account of the voluntary relaxation of the M. tensor tympani with marked lateral drum membrane displacement becomes distinctly related to the problem of middle ear mechanics and may be correlated with the function of the intrinsic ear muscles described by Wales.[38] The argument for independence of contraction in these muscles is presented by the latter in a cycle beginning with the swallowing reflex and ending in the swallowing reflex. The drum membrane position in the plus pressure phase (air injection) of the cavum tympani, brought about by tubal closure, is adjusted by a contraction of the M. tensor tympani. The minus pressure phase caused by air absorption gives rise to medial drum membrane displacement. This is resisted by stapedial contraction until a reflex is touched off causing salivation and swallowing. It is interesting to note that salivation was observed by Mangold in cases showing voluntary relaxation of the M. tensor tympani. It is unnecessary in the scope of this paper to go into details of which the readers are undoubtedly well informed. The writer is not in a position to disagree with the idea that air is actually injected into the middle ear by tubal closure. It has already been stated that the functional interrelation between the M. tensor tympani and M. tensor veli palatini, however probable, has not been established. It is clear that the contraction phase of the M. tensor tympani and M. stapedius, because of a limited drum membrane motion, might easily overlap at the mean of medial drum membrane displacement. The Wales and Politzer conception of muscle actions at that time would be identical in so far as the movement of the ossicular chain is concerned. Similarly Kato's synergistic action liberated through the auditory stimuli might again correspond in part to this overlap position in the two muscles. The reflex in the latter instance is auditory (inner ear) in origin, while in the former two it is perhaps a middle ear stimulus, or at least a reflex of different type.

We may liken the condition to corneal and retinal reflexes, both of which involve the lids.

Inasmuch as a muscle can do nothing more than shorten, it appears almost ridiculous that the function of these two muscles has not been thoroughly established, unless some other mechanical factor is present which has hitherto not been considered. If such a factor can be established, then çertain advances in the definiteness of information on the contraction of the two muscles may be anticipated. The old statement that the balance of the ossicular apparatus rests upon a balanced function of the muscles must be deleted as not in conformity with the facts.

Fifth: The bird appears to be able to shear off its fibrous external canal at will and protect its middle ear against violence from without in a manner unknown in mammals, with some exceptions. Further, the extreme of lateral drum membrane excursion meets a support in the application of the anterior drum surface to the external canal. The dorsal drum area is similarly protected by an erectile auditory pad—a wattle-like organ, found just lateral to the dorsal drum membrane circumference. The comparative physiology of this pad is interesting in that it is held responsible for the deafness in the "Auerhahn" (Tetrao) when the bird is "drumming" during the breeding season. The pad is separated from the tendon of the M. tensor tympani and from the external canal by a bursa. It contains stout elastic fibers which are put on the stretch when the pad is displaced lateralward either through congestion with blood or through lateral drum membrane (extracolumella) movements.

The mammalian drum membrane, however, by reason of its attachment to the more fixed malleus, the insertion of the M. tensor tympani, and its intrinsically heavier structure, is capable of very limited motion. In no extreme is the secondary support offered by the external canal, or by the medial wall of the tympanum or by the bracing of the cartilaginous extracolumella as found in birds. It appears, as might perhaps be expected from a study of the bird and its habits, that the air absorption and barometric variations are more readily compensated in this form than in the mammal.

Sixth: The bird possesses the two openings in the otic capsule which have already been mentioned in the frog. The fenestra vestibuli is filled in with the columellar footplate and an annular ligament strikingly similar to the relations found in mammals. The hinge action in the tilting of the stapes is, however, less pronounced. The fenestra cochleæ is somewhat larger in proportion to the footplate in birds than in mammals. In some forms (goose) this window is not directly related to the tympanic cavity but is placed in contact with the vena jugularis, and reminding one, in a way, of the extratympanic position of the membrana tympani secundaria in the frog. The relatively small round window of the mammal may be interpreted into terms of smaller compensations for mass displacement of the perilymph than in the bird. This is strikingly in keeping with a purely mechanical effect due to the peculiar differences in drum membrane mobility. The application of the membrana tympani secundaria to the vena jugularis is of first importance in the relation of intratympanic pressures to that of the perilymph and will be considered later.

Seventh: The bird, like the frog, possesses a passive traction system in relation to its middle and inner ear. The elastic ligaments appear to have a very definite office in limiting the amount of excursion of the sound transmitting apparatus —particularly its outward excursion. These ligaments also limit the thrusts of the sound transmitting apparatus upon the perilymphatic space. The elastic ligaments in the bird, which have been described in three sets, are placed to limit the lateral drum membrane excursion. They also adjust the columellar parts during this excursion and allow mass displacement of the perilymph with a minimum pressure development. The attachment of the footplate is distinctly elastic, as is also the membrana tympani secundaria at the round window. The latter may be regarded as a compensation opening in all forms. A large amount of drum membrane movement may directly affect the perilymph through the sound transmission system. The size of the membrana tympani secundaria may be considered in terms of displacement at the oval window to allow unusual mass shifting of liquid. Its relatively large size in birds and frogs may be readily explained on this basis, and similarly its small size in the mammal.

Finally: The entire middle ear apparatus appears from its ancestral history to be definitely set in for variations in drum membrane position and not in terms of tension. These variations in drum membrane position arise from two distinct causes on account of the closed tubes. They do not correspond to the causes of drum membrane fluctuation in the frog, where the tube is open and where air is actually injected during inspiration. The first of these causes is due to air absorption and the second to barometric variation. Both of these causes will result in drum membrane displacement, and neither, per se, has any very direct relation to the sound transmission problem.

It appears from the phylogeny that the mechanical requirements in the transference of sound vibrations in air to the watery medium of the perilymph are fulfilled with a dehiscence in the otic capsule (the oval window), and the development of a connection apparatus (columella, ossicular chain) from the catchment area (drum membrane). The appearance of the second opening (round window) seems to arise as an adjustment factor to a variable drum membrane topography. This variability in drum membrane position is primarily due to respiration in the frog with open tube, and secondarily to displacements arising because of air absorption or barometric variations in forms with closed tubæ. The sound transmitting apparatus may therefore be resolved into two distinct functions: the function of transmitting sound vibrations from the drum membrane through a solid medium (columellar, ossicular system) to the liquid of the perilymph; and second, an adjustment apparatus to allow for compensation in the system due to the variable drum membrane position.

A structural transition from bird to mammal occurs in the suppression of the quadratum; the formation of a definite annulus; the development of a three-part ossicular chain; the separation of middle ear complexes one from the other; a more immovable drum with a definite pars flaccida; a controlled tuba; the development of two muscles, a new M. tensor tympani, which arises out of the mastication muscles; and a persistence of a stapedial muscle comparable to the M. tensor tympani of birds. It appears that the ossicular chain preserves the old function of a columellar apparatus. Its bent-lever condition is more dependent on the factors developing the incus than those associated with either malleus or stapes (see later). That the contraction of the M. tensor tympani and M. stapedius have an effect upon acuity in hearing cannot be denied. The result may be in part central in terms of attention or actually damping on a mechanical basis. However, that this is the prime phylogenetic function is extremely questionable. Perhaps a comparison may render this more apparent—i. e., the difference between a prime phylogenetic or older function and a newer superposed function may be readily seen in the eye. It is known that narrowing the palpebral fissure increases the acuity in vision. The stenopaic slit eliminates astigmatic error, and a contraction of pupil sharpens the visual field. This result, however, is accidental in closing the lids. It is explained by the discharge of the normal sleep reflex associated with a relaxation of the M. levator palpebræ superior. The prime function of the lid is directly associated with maintenance of the watery medium for the cornea. It is adjusted by definite corneal reflexes which are distinct from the cycle to the lids from the retina—the reactions to light irritations. The retinal responses are again indicated in the same sleep reflex. To investigate the lid reflexes dependent on light and to disregard the corneal reflexes would be unscientific. To disregard middle ear function results because inner ear adjustments appear so self explanatory is not in keeping with the trend of modern investigation.

At the present time the actual transmission of sound vibrations through the ossicular chain is possible of definite physical investigation. There are widely contrary opinions on the function of the middle ear muscles and on the method of the registration of sound pulses at the end organ. It appears therefore that the possibilities in structural detail and the probabilities of phylogenetic significance must be seriously considered. The fact remains that individuals with malleus and incus gone may still hear, particularly through the telephone. They suffer a decrease in intensity of hearing for usual air transmitted sound, dependent largely on the size of the catchment area of the drum membrane in relation to a corresponding but inefficient area at the medial wall of the tympanic cavity.

The elastic character and function of the bird's middle ear ligaments has been overlooked for over half a century, as well as the definite sequence of columellar adjustments to drum membrane displacement. It may therefore be well to examine the middle ear region of the mammal to ascertain whether there are any other passive factors operating, apart from drum membrane elasticity, which are present in all forms. The problem of the elastic ligaments in the middle ear complex of mammals may be divided into two definite ones: first, the position of these ligaments, and second, their function. These are problems of almost indefinite length and much must be done, particularly in variable forms, before scientific conclusions may be attained. We present this brief preliminary survey to emphasize the line of thought to be followed. We report a confirmation of the theoretical presence of elastic ligaments in the baby cat as extremely stimulating toward some sort of defiinte solution—if a definite solution may be attained.

If we disregard aberrant aquatic forms, such as the cetaceans, the usual mammals may be divided into two very general classes based on peculiarities in the middle ear. The first variety, like man and the guinea pig, has a ligamentous support to the ossicular chain. In the second variety the malleus is attached by bony union of its processus anterior to the annulus like the carnivores (cat, for example). These differences do not seem to interfere at all with acuity of hearing, as both cat and guinea pig are possessed of excellent capacities in this respect. But the variations undoubtedly play some important part in middle ear mechanics which demand investigation.

The common cat is an animal with a remarkably well developed sense of hearing and differs from man in a number of particulars: first, a relatively long manubrium mallei is attached to the drum membrane, while the caput is relatively small; second, the bony union of the stout anterior process to the annulus is pronounced; third, the incus and stapes are relatively small; fourth, the M. tensor tympani is almost completely intratympanic in position; and fifth, the development of the bulla posteriorly, which may be disregarded, because it after all merely contributes to size of the tympanic space.

The fixation of the malleus is accomplished through attachment of the manubrium to the drum membrane; the bony ankylosis of the anterior process; a heavy reinforcing white fibrous ligament uniting mainly the anterior process to the annulus; the reciprocal incudomalleolar articulation which is sometimes three-part, sometimes two-part; and the attachment of the M. tensor tympani to an especially heavy muscular process which is not directed the length of the tendinous fibers but at an angle to them.

Two series of the entire middle and inner ear region of a newborn cat were cut in paraffin and stained with resorcin-fuchsin and differentiated in picric alcohol. The elastic connective tissue prepared with this technic is blue black, while the white fibrous tissue remains feebly stained. Examination of these two series reveals the following points:

First: The elastic fibers of the drum membrane are relatively poorly developed, as noted by Watsuji.[40] The membrane itself is made up for the most part of interlacing bundles of white fibrous connective tissue with few elastic elements.

Second. The ligaments attaching the malleus are of white fibrous tissue, which is particularly well developed in the attachment of the ankylosed processus anterior.

Third. The capsular ligament of the incudomalleolar articulation is elastic in character and the wedging in of the incus into the reciprocal articular surface of the malleus is very prominent.

Fourth. The posterior ligament of the incus attaching the short horizontal process to the posterior superior wall of the middle ear cavity may be definitely separated into two components: a superior lateral ligament of white fibrous tissue which has a wide superior attachment and whose fibers converge upon the superior surface of the short process; and an apical ligament which is directed downward and composed almost exclusively of stout elastic fibers. This observation on the elastic apical ligament in the cat (dog and guinea pig may also be included) confirms the work of Schmidt,[35] who described these two incudal ligaments in the human being.

Fifth. The capsular ligament of the incudostapedial articulation is also elastic in structure.

Sixth. The ligamentum annulare and the deep surface of the membrana tympani secundaria are made up of elastic connective tissue.

It appears therefore that all the ligaments associated with the incus and stapes are elastic in character except the posterior superior ligament of the incus. The apical elastic ligament of the incus in particular is suggestive because the incus is the passive bone of the series—i. e., it has no direct muscular attachments. Accordingly it lends itself well toward a comparison with the cartilaginous extracolumella of birds with its associated ligaments, particularly the columellar squamosal ligament of Platner, which has a pronounced function. Theoretically therefore the incus is a neglected factor in middle ear mechanism, and the presence of the elastic ligaments suggests passive factors of return of individual ossicle movements. This is in keeping with the recent investigations of Mangold, Kato and Wales, and contrary to the commonly accepted view of Politzer concerning the antagonism of M. tensor tympani and stapedius.

When we examine the method of fixation of the stapes footplate at the fenestra vestibuli and the character of the membrana tympani secundaria in the cat, we find a striking resemblance to conditions as found in the bird. The ligamentum annulare of the stapedial as well as that of the columellar footplate are composed almost exclusively of elastic fibers. These fibers are perhaps relatively shorter in the mammal, where the stapedial footplate movement is more limited and perhaps even mechanically inhibited from marked inward displacement. The membrana tympani secundaria is also formed by elastic fibers, particularly on its deep surface, and the structure of the membrane in cat and chick is surprisingly alike.

We are now prepared to consider the mechanical features in the Wrightson theory. We shall quote rather extensively from his recent work because it agrees with the commonly accepted conception of sound transmission. It disagrees only from other theories in the displacement responses of the membrana basilaris and the method through which the auditory cells are affected.

According to the Wrightson theory, we must look upon the cochlear mechanism as a weighing machine. That is to say, the membrana basilaris is an adapter membrane piston whose function it is to transform the vertical waves in the perilymph liquid into transverse displacements at the hair cells. It is not our purpose to offer objections to the extensive physical research in regard to the form of sound waves. We object rather to the function which is ascribed to the middle ear. We are not satisfied with the statement as to what is cause and what is result.in the interpretation of the sound transmitting complex.

"When the rarefaction part of the air wave advances on the drum membrane, the atmospheric pressure which exists in the middle ear drives the drum membrane backward." (Fig. 1.)

P. 50. "The malleus and incus are firmly attached to both the drum and the stapes, so that the fenestra ovalis is pulled outwards, and the atmospheric pressure of the middle ear through the fenestra rotunda also follows up the displacement caused by the outward motion of the stapes. We have thus power applied at the stapes to produce a positive to and fro motion in the liquids in the cochlea."

P. 54. "The greatest excursions observed at the extremity of the manubrium of the malleus were 0.76 mm., those of the long process of the incus 0.21 mm., and, according to Helmholtz, those of the stapes 1/18th to 1/14th mm., or a mean of 0.06 mm. These measurements are, however, the result of extreme condensations and refractions of the air in the external meatus or in the tympanic cavity. On the other hand, during vibrations caused by sound, the excursions of the stapes are limited (Gelle); and Reimann justly states that with weak but still plainly perceptible tones these motions of the stapes must be so small that they cannot be seen even with the most powerful microscope" (quotation from Buck after Politzer).

P. 56. "It is difficult to realize that these minute motions can be ordinary molar motions, but we have a proof supplied in the action of the phonograph. In this instrument the almost microscopic groove upon the wax of the receiver cylinder caused by the mechanical vibration of the one membrane is made to deliver up to a second membrane an almost exact imitation of the vibration of the first which produced the grooves in the wax. Molar motion is clearly the cause of the reproduction, and therefore it is reasonable to regard it as being the cause of the production."

P. 93. "We have now to consider two other membranes in the cochlea, the basilar and Reissnerian membranes, which form a closed triangular space (canalis cochlearis) running spirally from the base to the apex of the cochlea and enclosing liquid endolymph which entirely fills the triangular spiral space and cannot mingle with the perilymph, a liquid of different chemical constitution, which entirely fills the vestibular and tympanic passages. Any displacement of liquid occurring at the stapes can pass into the tympanic passage by the displacement of the Reissner and basilar membranes instead of going through the helicotrema, if the resistance is less through the former channel. Of course, no particles of liquid pass the membranes, but the pressure causes displacement into the tympanic passage, and this displacement proceeds onwards to the fenestra rotunda, where it gets relief in the middle ear, which is normally at the atmospheric pressure."

P. 95. "Thus the displacements of the fenestra ovalis, Reissnerian and basilar membranes, and the fenestra rotunda are equal, and the internal membranes move as though they formed part of the liquid."

P. 96. "It is evident that, taking into consideration the relative areas of the basilar membrane and the helicotrema, the line of motion of the displacements will follow that of least resistance, or through the basilar membrane, especially in the case of such rapid displacements as we have in sound waves."

P. 97. "If there were no basilar membrane, but a rigid partition, all the displacement produced at the fenestra ovalis would have to pass the helicotrema, and the reduction of area would cause a considerable loss of pressure in the tympanic passage owing to fluid resistances. With a flexible basilar membrane the displacement is passed through to the tympanic passage, distributing itself along the gradually widening membrane towards the helicotrema."

P. 121. "The stapes and basilar membrane move together because connected by inelastic liquid, which controls completely their movements. The stapes and drum move together because connected by an articulated chain of ossicles. Thus the pressure of the air wave on the larger area of the drum is passed through the bent levers of the ossicles to the much smaller area of the stapes. This increases the unit pressure in the liquid of the labyrinth like a small intensifying press, the increase of pressures implying a corresponding decrease of displacement and motion and therefore of velocity. The drum membrane transmits its energy through the ossicles in a direct chain of bones, so that as the varying pressures of the air wave arrive at the drum they immediately meet the resistance of the inelastic liquids in the labyrinth and act upon the combined resistances of the tensor tympani and stapedius muscles; also on the elastic connections of the basilar membrane and other resistances."

P. 46. "Dr. Keith points out that if the auditory human ossicles have to do with the regulation of the pressure of the perilymph and endolymph, the arrangement of the muscles in connection with the chain of bones becomes clear. In the first place, the two elastic muscles under initial tension are continually acting in opposite directions on the chain of ossicles to balance them. When the tensor tympani contracts it has the effect of plunging the stapes deeper in its fenestra and increasing the pressure of the fluids in the inner ear; the stapedius has exactly the opposite effect, and through the balance maintained by these muscles the pressure of the liquids in the inner ear is regulated."

Keith in the appendix of the book gives a rather exhaustive account of the ear relations in various forms, and inasmuch as he devotes some space to bird mechanics it is important to note his comments.

P. *222*. "The ear of the bird is likely to give us a clue to the mechanism of the cochlea for the following reason: All the evidence at our disposal leads us to believe that birds and mammals have been derived from a common ancestry; they have become very different in appearance because they have specialized along totally different lines. We feel certain therefore that the ears of birds and of mammals are modifications of a common form and that, although they may differ greatly in detail of structure, yet the underlying principle on which they work must be the same. For that reason I am to devote a few paragraphs to the ear of the bird. Retzius has published excellent sections of the bird's ear."

P. *221*. "Now, when we are in doubt as to the exact mechanism of any obscure structure in the human body, there is always one source of evidence open to us—that of comparative anatomy. To help us in deciding as to what may be the exact manner in which the cochlea converts sound impulses into nerve stimuli I am to refer to the cochlea of the bird for the following reasons: There can be no doubt the ear of birds has an analytic power; many of them can sing and have, we must suppose, the power of appreciating song; they distinguish one call from another. Several kinds of birds imitate human speech; such imitation would be impossible unless their ear appreciated and distinguished the various modifications of tone combined in our speech. We must therefore presume an analytical power in the cochlea of the bird."

P. *222*. "In the case of the bird, a single bone—the columella—connects the drum with the oval window. While the outer end of the columella is fixed to the drum, its inner end expands into a footplate which is fixed into the margin of the oval window by a ligamentous membrane. The lower and hinder borders of the footplate are more tightly fixed in the window than the upper and anterior margin, not unlike the manner in which the stapes is attached in the fenestra ovalis of the mammalian ear. The movements of the columella are more like those of a lever than of a piston; it is hinged to the lower margin of the oval window. Internal to the footplate is the cavity of the vestibule, filled with fluid. A horizontal partition is drawn across the floor of the vestibule, stretching from the lower margin of the oval window, which is occupied by the footplate, to the opposite or inner wall of the vestibule."

P. 223. "It will be noted that the middle passage (scala media) of the cochlea is separated from the cavity of the vestibule by a thick folded membrane containing many blood vessels—the tegmentum vasculosum. It represents a combination of Reissner's membrane and the vascular body (stria vascularis) of the mammalian ear.

The horizontal partition just described forms the floor of the vestibule and the roof of the lower or tympanic passage of the cochlea—at least the terminal part of that passage. The round window lies immediately below the oval window. It is closed by a strong but loose membrane, which is placed between the lower end of the tympanic passage and the cavity of the middle ear or tympanum.

Now, in considering the mechanism of the bird's ear we may omit from our calculations the movements of the tegmentum vasculosum; it is a slack membrane and may be regarded for our present purposes as forming part of the fluid which fills the vestibule. When, then, the outer end of the columella is set into vibration by the drum, its footplate will carry the impulses of the drum to the fluid filling the vestibule. As in the mammalian ear, we may distinguish four phases in each vibrational cycle. In phase I the footplate, starting from its point of rest, moves or rotates inwards, displacing a minute quantity of the vestibular fluid. The vestibule has firm walls everywhere except that part of its floor formed by the basilar membrane. The membrane yields, displacing the fluid contents of the lower passage and forcing outwards the round membrane. In phase II the footplate returns to its starting point, and the basilar membrane and organ of Corti rise to their equatorial level. In phase III the outward excursion of the footplate continues, and the basilar membrane rises so as to become convex upwards; the round membrane is drawn in. In phase IV all these parts return to rest."

There are a number of important features in the Wrightson hydraulic weighing-engine hypothesis of inner ear function which are clearly related to middle ear mechanics. They must therefore be considered from the standpoint of the causal factor—sound—in its behavior to the middle ear complex, and to the perilymph itself. It is essential that these points be clearly stated because Wrightson, an engineer of unquestioned ability, has interested himself in the problem for forty years, and Keith, who contributes the confirmatory evidence from an anatomic and physiologic standpoint, is an investigator of recognized attainments. If facts have been presented, we are dealing with differences in opinion regarding their interpretation. If, however, the facts have not been presented, then we are dealing with an attempt to make observed data conform to an hypothesis and may offer criticism which is entirely outside of differences in opinion or personal equation.

Briefly it will be seen Wrightson considers that the sound wave produces a molar or mass drum (see Fig. 1) membrane displacement. He believes that the ossicular chain functions as an "intensifying press which increases the pressure and decreases the velocity." The bent-lever transformer (ossicular chain) is regarded as balanced by two muscles—M. tensor tympani and M. stapedius, which may also have the office of acting as regulators of perilymphatic pressures. The pressure in the labyrinth is considered equal to that of the tympanic cavity (atmospheric) because of the relation of the stapedial footplate and membrana tympani secundaria to the tympanum. The membrana tympani secundaria is assumed to undergo displacement excursions dependent on the in-and-out movement of the stapedial footplate because the otic capsule is an inelastic container, excepting at the oval and round windows, and filled with an incompressible liquid. The displacement pressure caused by the stapedial movement is applied to its compensation area, fenestra cochleæ, through the membrana basilaris. This displacement arises because the resistance through this membrane is less than the friction head through the helicotrema. The membrana basilaris is therefore construed as a piston lying between the two openings in the cylinder—the oval and the round windows. The position of the Corti arches to the basilar membrane makes possible a tilting of the organ of Corti during the up-and-down movements of the membrane and results in a transformation of vertical movements into a lateral shifting of the hair cells at the tectorial membrane. The cilia would therefore respond to displacement forces applied at right angles to their length. The analyzing capacity of the end organ could in this manner be translated into a delicate weighing machine of pressures in the liquid.

Keith calls attention to the similarity in position of the essential parts of the inner ear system, notably in the bird, and also includes certain mammals and other forms of terrestrial vertebrates in his report. Inasmuch as the bird is a chief factor in his considerations, the relations in the bird may be also regarded from a somewhat different point of view later on.

We propose to ask several questions and develop in the answers to each one wherein the facts do not conform with the Wrightson assumptions. We will also show wherein the comparative anatomy and physiology fail to substantiate the Wrightson hypothesis.

I. Do the members of the ossicular chain act as an "intensifying press" which decreases the amount of excursion at the stapedial footplate in response to the drum membrane displacements occasioned by sound vibrations?

Practically all modern theories of hearing assume: (1) that sound vibrations produce a mass movement in the drum membrane; (2) that the ossicular chain acts like a lever transferring the membrane movements; and (3) that the liquid contents of the internal ear are displaced by mass stapedial reaction. There is, however, another method by which sound vibration may register an effect on the end organ of hearing. The writer wishes to discuss this method at some length.

Hence it will be necessary to consider the mechanical features of our problem rather fully. The accepted conception of ear mechanics is practically the one made famous through Helmholtz's work,[20] although Bell, and Bell[2] in 1812, presented the identical proposition, together with a figure to illustrate the leverage action of the ossicles.

Let us first consider the differences in the behavior of concussion and sound waves in air. When a gun is discharged two varieties of air displacement arise: a concussion wave, which is due to the mass movement of the air, and a molecular displacement in the air which transmits the sound of the report. The explosion wave travels rapidly from the source, but its velocity is decreased as it is diluted out in the distance, because of the resistance in the air which it must overcome. The velocity of the sound wave, however, does not behave in this manner and remains practically constant so long as the medium through which it is traveling is physically constant. The rapidity with which a sound wave is carried is dependent on the density-elasticity coefficient of the medium through which it is being transmitted. If one is walking on a railroad track, and repairers strike the rail one mile away (roughly, 1,600 m.), the concussion wave begotten in the rail and in the air will be practically, if not entirely lost, but the sound of the tap comes to the ear through the steel in about one-third of a second, while the air transmitted sound will arrive roughly four and one-half seconds later. This indicates that the wave length of a given sound through steel is about fifteen times the length of the same pitch in air.

This fact, however, does not bear any definite relation to the efficiency of the two media in respect to their capacity for transmitting sound. Speed, in other words, bears no definite relation to quantity, and the air presents a slow but well adapted material not only for the origin of sound but for its transmission as well. If one places a rod of glass and one of vulcanized rubber of equal length and thickness upon a resonator and applies the stem of the vibrating fork to one end, it is found that the resonator speaks out equally well. This occurs in spite of the fact that the transmission through glass is ninety times as rapid as that through vulcanized rubber.

The wave length of a given sound pulse in air therefore varies with its pitch, and is equivalent to the distance traveled in a second, divided by the number of vibrations. For this reason rapid motion toward the source of the sound will materially increase its pitch. It must also be remembered that air is a relatively slow transmitter of sound vibrations. The wave lengths in air, while they vary from 5 m. to about 3 cm., are short when compared with the waves of similar vibrational frequency in other media.

Sound is transmitted by a longitudinal molecular displacement, and its amplitude therefore lies parallel to the direction. The molecular displacements in a single pulse may be represented in four phases, each of which occupies the time of one-fourth of the frequency. The first phase is that of the molecular advance from a position of rest; the second is the retreat to the original position; the third is the swing-back, equal to the amount of the first motion; and the fourth is a readvance to the position of rest. This means that the sound vibrations travel by alternate rarefactions and condensations in the medium. It must also be remembered that during the first and fourth phases the molecular motion is in the direction of the wave propagation. In the second and third phases the molecules retreat as the sound advances. This is a distinct pendular motion and may only be represented graphically by translating the longitudinal displacements into transverse ones. It will therefore follow that the energy or intensity is quite independent of the wave length but not independent of the pitch. The higher the pitch, the greater will be the number of independent waves per unit of time. The rapidity of transmission is dependent on the medium, but independent of the power applied or the amplitude created.

The amplitudes or intensities of sound vibrations therefore represent the distances of actual molecular displacement which in ordinary sounds are of microscopic values, and, in the case of weak intensities, ultra microscopic measurements. Calculation has been made on the amplitude of an F sharp pitch when produced by a pipe actuated under an air pressure equal to a 40 mm. column of water. The amplitude of this sound vibration at 115 m. has been computed to be 1/25,000th of a millimeter. Let us compare the actual sine curve of this frequency and amplitude with that obtained from the oscillation of a tuning fork prong as recorded on a smoked drum. The tuning fork prong vibrating at a frequency of 181 per second will graphically reproduce the molecular behavior. The resulting curves will, however, be of transverse, not a longitudinal waves; second, the wave length will be tremendously increased; and third, the amplitude will be enormously exaggerated. The sine curve under the conditions indicated above consists in reality of a line 1.8 + m. long, with a deviation of 1/25,000th mm. Contracting the wave length to 1 cm. and magnifying the amplitude with a corresponding increase will only result in a deviation from the straight line of .0072 mm., or roughly the diameter of a human red blood cell.

The sine curve figures are pendular motions which graphically show molecular displacements. They are unquestionably correct if one remembers they represent transverse motions and not longitudinal ones. A misconception of the actual behavior of the sound pulse might readily come about on the basis of the sine curve figures produced by Wrightson and others. It has been estimated under the conditions noted in the F sharp pitch, at the distance of 115 m., an actual drum membrane movement would not occur above 1/1,000,000th mm., or, in other words, a mass movement in the membrane would probably not exceed 1/100,000th of its thickness. We are likely to visualize these alternating condensations and rarefactions in the air, due to sound vibrations in the form of explosion waves.

It is well to bear in mind that the oscillations of a tuning fork bear no relation to the wave length and the amplitude of the sound wave arising from this source.

It is usually conceded that the impingement of sound vibrations upon the drum membrane begets a mass response in the membrane. This may be illustrated by quoting from Daniel's[10] Textbook of Physics, from which the information regarding the F sharp pipe was taken. "Motion is thus communicated through malleus, incus and stapes; but the stapes moves only two-thirds as much as the handle of the malleus—another step in the increase of the force and the diminution of the amplitude of the vibrations conveyed to the ear by the air." The other factor mentioned was "that the handle of the malleus is wielded with considerable force—one step in the increase of force of the aerial vibrations on their way to the inner ear." It will appear that the mass movements in the drum membrane occasioned by increasing and decreasing the air pressure in the external auditory canal have been carried over directly to mass responses in the drum membrane without reference to the amplitude and force of sound vibrations themselves. Daniel, for example, offers on suggestion how a movement of 1/1,000,000th mm. at the handle of the malleus could undergo a one-third reduction in being transmitted to the stapes, nor does he mention the possibility of an elastic membrane absorbing a surface compression of 1/100,000th of its own thickness without an actual mass response taking place.

Wrightson has constructed a schematic working model to indicate the mechanics of sound transmission. This model differs in no essential particulars from the usual schemes except in a direct connection of the footplate of the stapes to the membrana basilaris. The motion in the drum membrane is actuated by a bent rod, clearly a mass displacement form. The ossicles are represented by a double lever with two hinges; one element, the malleus and incus, and a second hinge member, the tilting of the stapes, moved by means of the first. The muscles (M. tensor tympani and M. stapedius) are shown by two elastic bands which operate to balance the chain. The mechanical relation between the stapedial footplate and the basilar membrane and inner ear structures is also represented. While the scheme is merely presented for the purpose of visualizing the arrangements and motions in the various parts, several mechanical objections may be made. The first of these is the frequency with which the apparatus must respond; the second, the actual power applied to make it react; and the third, the distance through which the original activating factor must operate. We must consider not only the feasibility of the scheme but the minimum conditions under which it must function.

Let us take the same low frequency F sharp — 181 vibrations to the second and apply it to Wrightson's machine. A reciprocal apparatus which will respond to 181 individual impulses per second must indeed be delicately balanced, particularly when it represents a double lever of the form indicated. Carry the frequency to C_6 on the piano — 4,096 vibrations per second, well within the range of usual tone, and a mechanical motion of this kind becomes inconceivable in the form of a complicated hinge—give and take. The most delicately balanced supercentrifuge runs about one-tenth this speed and approaches a limit of physical possibility in the practical construction to withstand the centrifugal force.

It must be remembered that sound pulses are ordinarily of minute power value. Ten thousand people might readily hear the energy liberated by a single cricket. Yet one is asked to believe that this energy is sufficient to swing 20,000 sets of ossicles. Not only must the inertia of the mass be overcome but a relatively large amount of perilymph must also undergo shifting. Wien has computed that the pitch 1,600 requires the least amount of energy to register upon the end organ. He states this amount for an instantaneous tone is minus 1.10-15th erg. If Wien's figures are correct, then we may translate this energy readily once we know the weight of the ossicles and liquid contents of the cochlear system. Assume this weight to equal 125 mgs. The amount of energy required to lift a nickel five-cent piece 2 mm. from the surface of the table is the gram-centimeter unit. If minus 1.10-15th erg will move the weight of 125 mgs., then the gram-centimeter will move a block of concrete 200 feet by 50 feet and 35,000 miles long. The power applied by sound vibrations is therefore insufficient under normal conditions to move the mass, particularly when the entire system is aperiodic and a summation of energy impossible.*

It has been known for many years, and recently substantiated by Haldane (45), that the ear has an extremely fine interval discrimination. Haldane maintains that the trained ear can at certain pitches recognize the dropped pulse up to a frequency of 250th of a second. This deadstop in a rapidly swinging system makes it impossible to conceive a mass reaction because the inertia of the system must be considered zero. But this in reality cannot be the case. Wrightson himself assumes a factor of inertia because he calculates the resistance head to liquid displacement through the helicotrema is sufficient to divert the pressure from the scala vestibuli into the scala tympani through the vestibular and basilar membranes.

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*NOTE. Since writing this article Kranz has checked Wien's results, using the thermophone as a sound source under rigorous test conditions. He finds Wien's figures that it requires 100,000 times the energy to hear 64 than it does 1600 are essentially correct for 128 and 2048 respectively. The minimum rate of energy flow is, however, 10,000 times as great as that computed by Wien. The particle displacement at 2048 p.p.s. was 0,025 micromicrons or one-fortieth millionth mm.—Phys. Rev., Vol. 17, 1921, p. 384.

The writer cannot agree to the proposition that mass drum membrane displacements arise as reactions to sound vibrations of usual minute amplitude. We realize, however, that molecular forces may result in mass movements. The transformation of the sun's rays on the black surface of the radiometer causes a molecular bombardment of the air molecules of the partially exhausted tube. Here we are dealing with the effect of radiant heat, and the effect is an obvious movement of the vanes. There are limits to intensity of hearing just as there are limits to the reaction in rods and cones to light. When the amplitude of the sound waves becomes excessive and the drum membrane mass movements arise, we are undoubtedly well beyond our possibilities of intensity of hearing. In the writer's opinion the entire mechanism is such that the problematic mass movements in the drum membrane and in the ossicular chain cannot have anything to do with hearing.

Our answer, then, to the first question is this: In all probability no drum membrane displacements occur in the human ear under the usual conditions of hearing; nor is there any mass response in the ossicular chain and the perilymph. On the contrary, all such mass responses seem decidedly opposed to the efficient working of the apparatus. In birds no delicate lever system between the drum membrane and the perilymph is to be found. The movements in the columella occur only as a result of forces which are easily measurable. Many animals, moreover, of acute hearing (cats, for instance) have a bony ankylosis of the malleus to the annulus.

II. Is the antagonistic action of the M. tensor tympani and M. stapedius responsible for a balance in the ossicular chain?

The comparison of these two muscles with two elastic bands is unobjectionable. It is, of course, a mere figure of speech intended to make certain mechanical relations clear to the uninformed. But we must question the assumption that these two muscles act in opposition. This assumption was evidently made without reference to the recent detailed and wonderfully graphic work done by Kato and by Mangold. In the light of this work the assumption of Wrighton becomes untenable as far as the mammalian ear is concerned. Birds also enjoy a fine sense of hearing and have only one muscle associated with their sound transmission system.

It was pointed out that Mangold's researches on cases of voluntary relaxation and contraction of the M. tensor tympani in man do not harmonize with the older Politzer conception of antagonism. It was also stated that Kato's excellent observations in the cat and the rabbit deduce a synergistic action in these muscles. Further the contraction of the muscles is in part a response to prolonged auditory stimulation. This must necessarily be the case, because the reflex system is too slow to be operative in terms of speed of sound itself. It may be that a serious misconception of the action of these muscles has come about. This misconception may be due to translation of drum membrane movements into terms of usual sound wave reactions, and from observation of the muscles under certain conditions without reference to other possibilities and probabilities. Finally there are certain unconsidered factors which may affect the middle ear mechanics—the presence of elastic ligaments.

We may therefore answer question II. The assumed antagonistic action of the M. tensor tympani and M. stapedius is not a fact except in a very restricted way. The alleged balance of the ossicular apparatus through muscle contraction in the manner described by Wrightson still remains to be demonstrated. Wrightson grants a balancing of the ossicular chain through the opposed action of M. tensor tympani and M. stapedius without suggesting why this is necessary. He does not even indicate what the disturbing element in balance may be. Why may not the ossicular chain be considered already balanced without muscular pull at all? The complete answer to this question will be found at the end of the paper when the comparative anatomy and physiology of the middle ear region are discussed.

The balance in the ossicular chain must logically affect the tympanum because of its close tuba. This brings up the question of the intratympanic pressure, the perilymphatic pressure and the relation of these two, because Wrightson maintains there is a distinct relation between them. The first part of this question may be considered presently; the second will be deferred until the perilymph container has been discussed.

III. Is the intratympanic pressure equal to that of the atmospheric?

Intratympanic pressure is conceived by Wrightson to be atmospheric. This is clear from the excerpts given and from his figure. He furnishes, however, plenty of evidence why it should not be considered equal to atmospheric pressure. It is well known that any moist vascular area exposed to an air pressure higher than that of blood-lymph tension may absorb oxygen, provided the surface membrane is permeable. It is also well known that air is absorbed from the middle ear cavity, indicating tubal insufflation as a remedial measure in cases of tubal closure. That some pressure differences may occur is indicated by Wrightson. For he grants a reflex cycle: "Swallowing or yawning causes a slight opening of the eustachian tube, which restores the equilibrium." He also appears to assume that this equilibrium is adjusted by a middle ear function because "increases in pressure in the middle ear due to bending of the drum membrane between the attachment of the malleus and the periphery of the drum might cause a slight rise in pressure in the middle ear, but such increases would not be transmitted to the stapes, as the same rise in pressure acts through the fenestra rotunda, thus balancing that acting on the fenestra ovalis." The contention of Wrightson that the middle ear is at atmospheric pressure is open to serious objection. He appears to show the pressure is never that of the atmosphere except at the time of tubal opening. He also seems to indicate that perilymphatic pressure may only be atmospheric at this time because the intratympanic pressure directly affects the perilymphatic pressure through the two windows (fenestra vestibuli and fenestra cochleæ). How a reflex mechanism can be touched off without a cause is in this instance not explained.

Wrightson's contradictory statements practically furnish an answer to question III. Intratympanic pressure may not be equal to atmospheric pressure except when the tube is open. The explanation of the inferred relation of intratympanic pressure to that of the perilymph will perhaps make the answer to this question a little more obvious.

IV. Is the otic capsule, filled with liquid and membranes, to be considered an inelastic container apart from the fenestra vestibuli and fenestra cochleæ, and what relation have these two windows in preserving a balance of perilymphatic pressure which Wrightson assumes to be that of the atmosphere?

The answer to this question is extremely important, because upon its affirmation rests the entire Wrightson hypothesis of an hydraulic weighing engine. The membrana basilaris is assumed to act as a piston responding to the force transmitted by the stapes, because of the relation of the footplate to the membrana basilaris and to the fenestra cochleæ. It may be well to discuss this answer more fully because of the importance in relation to this, as well as any other theory or hypothesis connected with inner ear mechanics. We may disregard what might be termed a quibble and assume that the highly elastic petrosum is inelastic in so far as the minute power applied to the perilymph from the sound reactions of the stapes is concerned. There are, however, a number of objections which Wrightson as an engineer might not realize. But Keith, his adviser for the anatomic and physiologic aspects, either did not know these objections or he disregarded them. The criticism of the Wrightson hypothesis of a weighing analyzer must not be made on its theoretic possibilities but on its workability under the conditions imposed.

It has already been granted that the petrosum is relatively inelastic or incompressible in so far as the power of the sound vibration applied is concerned. For otherwise it would be impossible to analyze the Wrightson hypothesis at all. We assume, then, an inelastic container. A minute stapedial movement, according to Wrightson, will displace the basilar membrane like a delicate piston, because this membrane offers less resistance than the friction to a perilymphatic flow through the helicotrema. A displacement of the basilar membrane must occur because of its position, situated between the stapedial footplate and membrana tympani secundaria.

Keith states definitely that the otic capsule may be regarded as inelastic apart from one point which he makes in connection with perilymphatic pressure regulation. It is difficult to determine why these writers accept the work of Bezold[5] on the molar properties of the sound wave at the stapedial footplate and completely ignore the exhaustive researches started by this writer in 1880 and completed in 1908 in reference to this particular problem. Bezold's work is not only typically careful in the matter of measurements, but it is checked by confirmatory evidence with a labyrinth manometer. Bezold is not convinced that the membrana tympani secundaria has any definite response to sound vibrations in the perilymph. His experiments in the reactions of the membrana tympani secundaria to variations in intratympanic pressure indicate a measurable fluctuation in this membrane without a response at the stapedial footplate. This, according to Wrightson, may not occur. These fluctuations of the membrana tympani secundaria are due to areas of less resistance than that at the stapedial footplate, in the aqueductus vestibuli, aqueductus cochleæ and porus acusticus interna. Pressure at these areas gives rise to variations in the perilymphatic pressures, as is evidenced by the labyrinth manometer. Bezold not only indicates the minute blood vessels as direct adjustment factors but even includes the lymphatic system. This evidence explains the failure in response of the stapedial footplate to the measurable variations in position of the membrana tympani secundaria with an equal negative or positive pressure applied to both orifices. Bezold's arguments for holding that the membrana tympani secundaria does not respond to sound vibrations appear reasonable. The only mention which Keith makes of all this work is to say that the minute capillary adjustments may be "disregarded as a part of the liquid which fills the cavity." This in spite of his notation on perilymphatic pressure—"The tension cannot be greater than that within the blood venules of the inner ear; it cannot be greater than venous pressure. The pressure would thus vary with the venous pressure; we may infer it would support a column of mercury two to four millimeters high."

Objections might suggest themselves to the experimental evidence submitted by Bezold on the basis that his results were obtained on dead material. These objections may not obtain for the work of Kato, who analyzed this very point on living material. "The reflex contractions of the M. tensor tympani may result in a medial displacement of the ossicular chain.

At least it follows from observing the anatomic relations that if the drum membrane is drawn medialward through contraction of the M. tensor tympani, the entire chain of ossicles would also move in this direction. This movement would necessarily be carried over to the liquid of the inner ear, and the result of muscular contraction would therefore evidence itself in a motion at the round window. A number of experiments were undertaken to answer this question—whether or not movements at the round window could be observed during the reflex contractions of the M. tensor tympani. The fluctuations would necessarily occur in a very restricted region and might be observed with a Westien magnifier in the behavior of the light reflected from the surface of the membrane. It was demonstrated in every instance that, at least with this magnification, no displacement of the membrana tympani secundaria could be observed as long as the intrinsic muscles were intact. Convexing of the membrane toward the tympanum was, however, demonstrable when the tendon of the M. stapedius was severed or that muscle incapacitated through destruction of its nerve. Cutting the M. tensor tympani eliminated this movement."

We read some time back that perilymphatic pressure is atmospheric and that it is regulated by the apposition of the two windows to the tympanum. Therefore when the pressure falls or rises in this cavity there is no displacement at the stapes or round window because they are both applied to the tympanum. Bezold's experiments, however, clearly indicate that this is not true. Keith himself states that the perilymph pressure varies with the venous pressure. He also disregards the capillary system as an adjuster of the pressure in an equally emphatic manner. This may account for the interesting statement made by Wrightson that "according to Dr. Keith, if the auditory ossicles have to do with a regulation of pressure in the perilymph and endolymph, the arrangement of the muscles with the ossicular chain becomes clear." This would indicate the definite conviction that a regulation of perilymphatic pressure by the muscular action is quite obvious. Wrightson's statement implies further that if we do not assume this to be the end result, the function of the intrinsic muscles is not clear. Keith's convictions in this respect, however, are not as strong as one might anticipate. Toward the end of his contribution to Wrightson's book we observe the following passage: "The tensor tympani *could* press the stapes inward and raise the pressure within the ear; the stapedius *could* produce an opposite effect; between them they could regulate the pressure of the inner ear—*within certain limits.*" Italics are mine to indicate the sentence should end with an interrogation mark and not a period. The problem of perilymph pressure necessarily also arises in birds, and certain features in comparative anatomy and physiology must be considered.

Our answer to the fourth question is: The otic capsule is not an inelastic container, either theoretically or practically; and the two windows are not the only functioning openings in this capsule. From the determined fluctuations in the membrana tympani secundaria without corresponding result at the stapedial footplate when equal plus or minus pressures are applied intratympanically, we may infer that minute stapedial movements are compensated for elsewhere. This compensation takes place through adjustments in the venous pressure for which there is direct evidence, as will be shown later. We must therefore take up the consideration of the physical reactions in the membrana basilaris and the directed force of the minute stapedial displacements on this membrane as opposed to all other pressure adaptive areas.

V. May the membrana basilaris be considered a transformer of vertical into transverse displacements because of its relation to the stapedial footplate and the membrana tympani secundaria, and because the route through the basilar membrane offers less resistance than liquid displacement through the helicotrema?

It will be recalled that the piston action of the membrana basilaris is the keynote of the Wrightson hypothesis. This piston action supposes an inelastic otic capsule with only two areas of possible surface motion, the stapedial footplate and membrana tympani secundaria. The experimental evidence of Bezold, however, is quite contrary to this assumption. Bezold holds that the fluctuations of the membrana tympani secundaria with a closed tympanum creating both negative and positive pressures at both windows are readily observed without stapedial movements. The fluctuations of the membrana tympani secundaria, accordingly, imply other regulatory factors, namely, those already presented in the foregoing answer. It was also noted in this answer that Keith himself believes the pressure is a variable one and dependent on the pressure in the blood vessels. The Wrightson hypothesis calls for a directed pressure response in the membrana tympani secundaria and for a piston action of the basilar membrane because of its relation to this orifice. The theoretical behavior of the liquid displacement at the helicotrema, while carefully studied by Wrightson, therefore, is dependent on hypothetical premises which are not in keeping with the facts. It may therefore be disregarded in spite of the careful calibrations and painstaking calculations. The ascribed motion of the membrana basilaris in a definite phase of responses to stapedial movement is one possible manner of creating transverse movements at the auditory hairs. The transverse movement, however, is neither a theoretic necessity nor a demonstrable fact. There are other methods by which transverse movements at the hair cells might be produced if it is considered essential that such movements actually occur or are necessary. The answer to question V may be stated as follows:

The Wrightson hydraulic engine therefore becomes one with an elastic cylinder and many theoretical pistons, and therefore many hypothetically directed possibilities. The objections are, however, sustained and must be controverted before we can accept the Wrightson hypothesis. To us this hypothesis is nothing else than an ingenious interpretation of facts as they appear to Wrightson but not of the facts as they actually occur.

The curious mechanical relation of the middle ear to the inner ear calls for the solution of another problem to which we must now turn our attention. The problem is:

VI. In what manner does the Wrightson piston movement of the basilar membrane occur when the sound vibrations are transmitted through the temporal bone to the inner ear?

While Wrightson has considered this point in the presentation of his evidence, the chief exposition was left to Keith, whose explanations are quoted in extenso. These explanations occupy the two pages on the bone conduction of sound vibrations. "There remains one very important matter which must be considered because of the light it throws on the mechanism of hearing. We have been regarding the stapes as the main agent in producing the fluid displacements which stimulate the organ of Corti. There are on record authentic cases of men and women who have been able to hear after the footplate of the stapes has become immovably fixed in the oval window. So long as the organ of Corti is intact and the round window flaccid, hearing is still possible. There is another allied condition. Patients who have become partially deaf from destruction of the drum of the ear have their hearing improved when a slight resistance, such as a pellet of cotton wool, is applied to the stapes. When the movements of the stapes are hindered hearing is improved. When sounds are conducted to the inner ear by means of the bones of the skull, in people with normal hearing, the volume of sound is markedly increased if the movement of the stapes or drum is hindered. I do not think these conditions have been yet explained."

"How are displacement volumes produced under direct bone conduction? The total capacity of the cavity of the inner ear of man I estimate to be approximately 200 cubic millimeters; 130 of these are contained in the main cavity, or vestibule and semicircular canals; 70 are contained in the passages of the cochlea. I presume that, were the thick, exceedingly dense and elastic bony walls of the inner ear completely closed, the fluid filling it would be subjected to a rise of pressure during the condensation phase of every sound wave; the pressure would fall in every rarefied phase. If the cavity, instead of being completely closed, had a flaccid window in it, then the rise and fall of pressure would be accompanied by a movement of that membrane. I presume that in the condensation phase of a sound wave a column will be displaced from the vestibule towards the round window; in the rarefied phase it will move back towards the vestibule. The basilar membrane will participate in all movements of the displacement column and give rise to stimulation of the hair cells. If, however, there are two windows in the inner ear, the oval one in the wall of the main chamber as well as the round window in the lower passage of the cochlea, part of the displacement column will escape by thrusting the stapes outwards; the part which thus escapes is ineffective, so far as the basilar membrane is concerned. Fixation of the stapes prevents such an escape. We see, then, why hearing is improved, in cases of bone conduction, when the movement of the stapes is hindered. We also see how small must be the volume which gives an effective movement of the basilar membrane. We have thus in direct bone conduction a means of discovering the actual volumes required to produce effective movements of the basilar membrane. I am certain that von Helmholtz greatly overestimated their size. He supposed that the length of the ligaments which bind the footplate of the stapes to the oval window might be regarded as indicative of the excursional movements of the stapes. The lengths of these ligaments represent the limits of stapedial movements—the limits of the movements produced, not by the drum of the ear, but by the two muscles which act on the footplate of the stapes. The actual figures given by von Helmholtz for the inward movement of the footplate in a complete excursion were 56 to 73 mm. The main cavity of the inner ear contains 130 cubic mm. of fluid; how much is displaced when a sound wave of a known vibrational rate and amplitude passes through the temporal bone? If we knew that, we could estimate the actual displacements of the basilar membrane and the movements of the hair cells when such and such a note is sounded. I think it will be found that the effective stroke of the stapes is less than 25 mm. in even the loudest tones."

The sound pulse in advancing through the temporal bone is said to increase the perilymphatic pressure during its condensation phase and to decrease the perilymphatic pressure during its rarefaction phase. The displacement of the liquid will therefore take place at both windows, thrusting them outward into the tympanum. This will naturally decrease an excursion of the membrana basilaris. However, if the stapes is held fixed, then all displacement thrust, according to Wrightson, will pass through the basilar membrane. The increased membrane reaction might therefore account for the resulting condition of increased efficiency in appreciation of sound through bone transmission.

There is clearly something amiss in this conception, because the entire Wrightson hypothesis is based on an inelastic container as mentioned before. Unless we assume this inelasticity in the container in reference to the power and frequency of sound vibrations, we cannot even consider the hypothesis at all. However, when the hypothesis is applied to hearing due to bone transmission, then the container (petrosum) at once becomes an elastic capsule, because the liquid-membrane filler is incompressible. So elastic, in fact, does the container become as to participate in the rarefactions and condensations produced by the minute and rapid sound vibrations. How the container may be regarded as inelastic in one part of this hypothesis and elastic in the other is not clear, to put it mildly. Nor is it intelligible why the liquid and membranes may be considered incompressible in one aspect of the problem and compressible under another, because the intralabyrinthine pressure varies with that of the venules.

Keith adds that if we knew "how much is displaced when a sound wave of known vibrational rate and amplitude passes through the temporal bone," "we could estimate the actual displacements of the basilar membrane and the movements of the hair cells." This statement gives the impression that we are dealing with scientific probabilities, when, as a matter of fact, we are dealing with physical impossibilities.

According to Keith, "Everyone who has studied the mechanism of hearing has presumed that the walls of the inner ear are rigid and closed excepting at the two fenestræ." He adds: "In that I think we are justified." In spite of this we are asked to believe that a diffuse vibration in the temporal bone may give rise to variations in perilymphatic pressure.

Our answer to question VI may be stated briefly: The piston action of the membrana basilaris cannot take place as a result of bone transmission. The evidence at hand does not disprove the point that sound is normally transmitted through the drum membrane and ossicular chain, even when the stem of the vibrating fork is applied to the vertex.

VII. Wherein does comparative anatomy show a confirmation of the Wrightson hypothesis?

The answer to this question is very simple and very brief. If one assumes that the membrana basilaris functions as a piston because of its position between the oval and round window, then comparative anatomy confirms this assumption with regard to amphibians, reptiles, birds and mammals. If we assume that the membrana basilaris does not function in the manner described by Wrightson then the position of the basilar membrane determined in the amphibian holds for all the other vertebrates on the basis of structural pattern, and the acuity in hearing is in no way affected. However, in the case of the birds a very serious objection may be made. It is more serious than in the case of the mammals, because the tegmentum vasculosum, corresponding to Reissner's membrane, lies at the columellar footplate aspect of the basilar membrane and forms a delicate and highly vascular shield. Now if the blood vessels must be considered highly efficient regulators of perilymphatic pressure, as Bezold has suggested and Keith has all but admitted, then the directed pressure of the columellar thrust must pass through a very specialized absorber before it displaces the membrana basilaris at all, not to speak of the membrana tympani secundaria. Keith regards the tegmentum vasculosum as part of the liquid which fills the cavity. In the goose the secondary tympanic membrane is not even related to the tympanum but is applied to the vena jugularis. The goose therefore has but one opening—the fenestra vestibuli—applied to the tympanic cavity, and the pressure exerted by the air of the middle ear is not effective on the membrana tympani secundaria. The pressure displacement of the perilymph in this form must exceed that of the small blood vessels of the extensive tegmentum vasculosum before the membrana tympani secundaria may become operative in adjusting the mass displacements.

It is noteworthy that Keith in his appeal to comparative anatomy makes no mention of the pressure regulation factors in forms with a single muscle (amphibians and birds), although it was suggested that the antagonistic function of M. tensor tympani and M. stapedius in the mammal were related to this factor.

It is true that careful measurements and detailed histologic investigations have been made by Wrightson and by Keith on the relations of the membrana basilaris and the position of the organ of Corti, but this valuable material has no relation to the problem in hand: the Wrightson hypothesis. We, therefore, cannot agree with Keith when he says with reference to the birds "that the theory put forward by Sir Thomas Wrightson, if it does not give us a full and complete clue to every item in its structure, serves this purpose to a much higher degree than any other I know of." We should rather say: The Wrightson hypothesis when applied to the birds does not work and fails utterly when applied to the goose, which is remarkable for its acute sense of hearing.

It would appear that the investigators following the mass displacements are not considering other possibilities. They accept as fact the sequence of events suggested by Helmholtz and therefore do not feel called upon to justify them.

Beyer[3], after a very complete study of the anatomy of the middle ear region in practically all vertebrate forms, makes very positive notations that the Helmholtz conception of a mass reaction in the ossicular chain cannot obtain. In particular, the leverage system reducing the amplitude of drum membrane vibrations meets with many serious objections. Forms in which the ankylosis of the malleus to incus and the ankylosis of the malleus to the annulus is the rule show no diminution in the acuity of hearing. The analysis of the anatomic relations of the incudomalleolar articulation by Frey[15 16] on complete serial sections of fifty-five species of mammals also substantiate this very point.

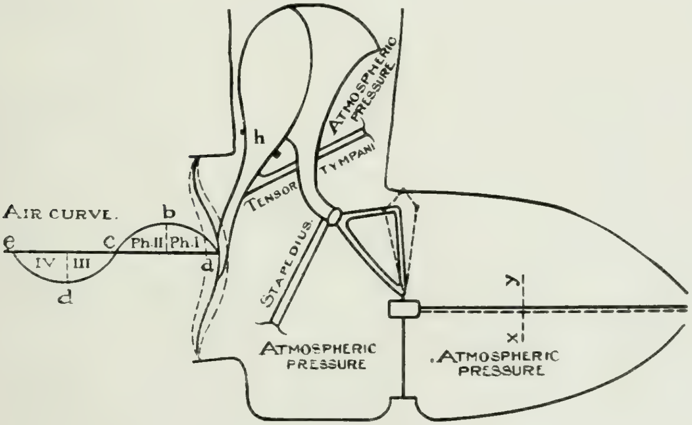

Figure 1.

II.

INTRANASAL RECONSTRUCTION.

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By reconstruction we mean the returning of the nose to as near its normal condition as possible, with the least amount of damage or destruction to its tissues.

The nose is the protector of our lungs by cleaning and modifying the air currents passing through it. Therefore, the size of the openings in the two sides should be as nearly equal as possible, with all the turbinates in place and the mucous membrane in normal condition, if we are to get the utmost use of these structures.

Opening into the nose are various sinuses: frontals, ethmoidals, sphenoidals and maxillary, which must have drainage and aeration, properly obtained only with a normal nose. The nasal passages are really the continuation of the eustachian tubes, and normal hearing is dependable more or less on a normal nose. In the past we did not fully realize the function of the nose; and still at the present time too much normal tissue is being removed which not only takes away our protection but leaves nonfunctioning scar tissue.

With a normal nose in mind and ever remembering its function, we examine to see if the air chambers are equal in size, and if not, what makes the difference. Is the septum straight, thin and in the center line, or is it bent to one side, or is it straight but thick between the middle turbinates, or has "spurs" of various sizes? Are the turbinates hypertrophied or hyperplastic, stand out from their attachments, are bulbous or have hyperplastic ends and borders? Is either the septum or turbinates or both interfering with the drainage and aeration of the sinuses or the aeration of the eustachian tubes?

A deflected septum is to blame for most of the troubles in the nose. It is not only out of line itself, but also causes pressure atrophy of the turbinates on the one side; and nature, in trying to equalize the air currents, enlarges the turbinates on the opposite side, and with these bulgings, thickenings, hypertrophies and hyperplasias, the air is not properly filtered and modified and the drainage and aeration of the sinuses are interfered with.

SEPTUM.

In the reconstruction of the septum, the submucous resection operation only should be done. Cutting or sawing operations do not straighten the septum and leave large scar areas which have no secreting function. In a submucous resection the only damage to the mucous membrane should be a short, threadlike scar at the anterior nares on one side only. A complete submucous resection should be performed in every case. As much of the cartilage as possible is to be left; this supports the cartilaginous portion of the nasal bridge and prevents the flapping of the membrane when the nose is blown. The bony septum should be taken out high enough up to prevent tenting of the membrane and to allow it to hang not only straight but in the center line. The crest must be well taken out to prevent the tenting below. Too many operators, especially beginners, do not observe these points. Incomplete operations and the tenting above and below are to blame for failures. We hear of too many operators doing a submucous resection against time. I care not who the operator is or what his experience, he cannot do a complete operation and with the least damage to the flaps, when time only is considered.

The cartilage and bone between the middle turbinates must be well removed, as the air currents in breathing pass this point and the drainage of the frontal and anterior ethmoid sinuses is affected by it. In nasal troubles an apparently straight septum is generally thick and should be operated on for this reason. If, at the time of the operation, both flaps are torn, a not too large perforation can be closed with a sliding flap. If this is not possible and the hole is opposite the lower turbinate, the turbinate can be used to close it. In nearly all old perforations, if not too large, the lower turbinate or portions of it can be used to close the hole.

After an operation, if a patient has a small perforation anterior which "whistles," more of the cartilage in front of the hole should be removed to make the septum straight. The whistle is due to the ballooning out of the membrane by unequal air currents. In some cases it may be necessary to enlarge the hole. A month or two after a submucous operation, if the patient is unable to breathe easily, the soft hypertrophies on the septum anterior to the ends of the middle turbinates are to blame and should be cauterized. If the membrane is lax and flutters when breathing, it can be tightened by cauterizing the thicker areas high up and back.

TURBINATES.

In reconstruction of the turbinates, none of the functioning part is to be removed. After a septum operation, if given time, nature will in nearly every case reconstruct the turbinates. The small ones will become large and the large ones small, owing to the air currents passing over them. Occasionally, it will be necessary to help nature. If they stand out too far they can be broken down, and if they are in too close they can be fractured out.

On the lower turbinates all hyperplastic posterior ends or lower borders are to be cut or snared off, but the bone is not to be injured. Occasionally it will be necessary to cauterize stripes through a persistently hypertrophied lower turbinate to reduce its size. Portions of the lower turbinate are not to be removed when operating for antrum disease; besides its other uses, it helps to protect the new opening from dirt and cold.

On the middle turbinate the hyperplastic posterior end and lower border is to be cut or snared off and the enlarged lobulus on the anterior end is to be removed. If the middle turbinate has a large bulbous or cystic end, the lateral half of the cystic wall is to be removed, it being lined on both sides with mucous membrane. A thin turbinate is the result as soon as the wound heals.

In operating for frontal, ethmoidal or sphenoidal disease, care is taken not to remove any of the middle turbinate, all of the work being done under it. The cavity is treated until lined with healthy mucous membrane; the turbinate is then broken in to help fill the space if necessary.

So far reconstruction in atrophic catarrh is in the experimental stage. As the ethmoids are involved, these cells should be opened and made into one large cell with perfect drainage under the middle turbinate. As soon as the cavity is lined with mucous membrane most of the secretions and crusting will cease. If the septum is badly deflected a submucous resection should be performed to equalize the air space. The crusts are kept soft with oily lotions. The whole mucous membrane surface is stimulated with applications like iodin-glycerin, and a hand suction pump used every day for a few minutes to draw blood into the membranes.

Working along the line of endocrine disturbances we have been giving suprarenal gland tablets, 1 grain three times a day, over a long period of time. Not only the nasal but also the patient's general condition is improved. After the secretion is somewhat stopped and the membrane becomes thickened some building up can be tried like that suggested by Dr. Beck, using pieces of bone under the mucous membrane.

SUBMUCOUS RESECTION.

The patient is operated on in the semireclining position. He is then comfortable, does not faint, there is better control of his head, and the operator is working with his arms down in a natural position. Nature has wisely provided a coating to the mucous membrane of the nose and an immunity to the germs of the air; therefore, it is better to do very little cleaning before a submucous resection for fear of breaking up this immunity. The outside of the nose is cleaned with alcohol and the face is covered with a piece of gauze moistened in 1:5,000 mercuric chlorid solution. The hairs at the opening are cut to give a better view, and the inside of the vestibule is wiped with iodin followed by alcohol. Both sides are sprayed with a little cocain (4 per cent) and the nerves blocked by the following method: The ends of four wooden applicators are slightly notched to prevent slipping and wound with a small piece of cotton. The cotton is then moistened with epinephrin (1:1,000) and applied to flaked cocain until it becomes saturated.

One applicator is placed as high up as possible on a line with the anterior end of the middle turbinate which blocks the anterior ethmoidal nerve; another is placed at the posterior end of the middle turbinate and blocks the sphenopalatine ganglion. This is repeated on the other side, as it is necessary to block the nerves on both sides of the septum.

A piece of cotton, saturated with epinephrin (1:1,000) is placed against the septum on each side to prevent bleeding during the operation. The patient is then given a hypodermic of ⅛ grain of morphin with 1/250 grain of atropin. This small dose does not make the patient "dopey," but it is large enough to cause relaxation and to take away fear; it also helps to prevent the after bone pain when the cocain wears out. In about twenty minutes the cotton and applicators are removed and the operation is performed without pain and, in most cases, with very little bleeding.

The nostril is held open with a Monosmith or right angle nasal speculum. The cut is made with a Freer septum knife on the convex side of the deflection; it is thus possible, as the membrane is elevated, to lift out the flap and so get around the bend without tearing it.

The cut is in the mucous membrane at its junction with the skin and extends from the top of the nose down to but not upon the floor, and goes through the mucous membrane and perichondrium to the cartilage. The membrane is then pushed back at the center of the cut with the edge of the knife until the white, shining cartilage shows. The membrane raises easily if the elevator is between the perichondrium and the cartilage, and with difficulty if between the perichondrium and the mucous membrane.

The membrane is then raised for a short distance with a small elevator; the right angle elevator is now used to elevate from the inside toward the cut, both above and below the original elevation. The original incision is now completed with the knife through the length of the membrane, cutting from the inside out, both above and below. In starting the elevation this way it is possible to avoid cutting through the cartilage and the membrane on the other side if the original cut is extended down to the cartilage through its whole length. The membrane is now raised with a long blade elevator as far back and as low down as possible or, if there is a spur, to its sharp edge. The original cut is now extended to the floor; bleeding comes with this cut, and so it is made after most of the membrane is raised, to have a field clear of blood while starting the elevation.

The floor is then elevated with the small elevator; it starts with some difficulty, as the membrane is bound tight to the bone at this point. After the floor is elevated the membrane on the septum is raised from below to meet the elevation from above or, if a spur is present, to elevate the under side up to and freeing its sharp edge.

In many cases it will be necessary to use a knife to cut the strong adhesions between the membrane and the bony septum where the crest of the superior maxilla joins the cartilage and the perpendicular plate of the ethmoid, as the fibers of the perichondrium and periosteum cross here.

After the membrane is completely raised it extends from the top of the nose out on the floor like the side of a tent.

The cartilage is incised with a Freer original septum knife just inside the original cut at an oblique angle, which brings it through at a slant just under the perichondrium on the other side. As much of the mucous membrane as possible is raised on this side, being sure to elevate well down on the floor in front with the right angle elevator, as this is the place where the chisel will enter later.

The Foster-Ballenger speculum is now inserted and holds the flaps of mucous membrane away from the cartilage and bony septum. The flaps are then inspected to see if they are entirely free.

After the flaps are elevated a small slice about one-eighth inch wide is taken off the cartilage along the inner edge of the cut. This is to prevent exposure of the cartilage if the flaps retract in healing. To avoid the dropping of the cartilaginous bridge and give it support we leave in place as large a piece of cartilage as possible, the thickness and deflection determining the size. A nick is made in the cartilage with the scissors as low down as necessary to retain the piece of cartilage desired. The Ballenger swivel knife, starting in this cut, is carried in and up with a half curve, then in, then down and then out, following the cartilaginous bony union. This cut piece of cartilage is now removed. The piece of cartilage left is easily pushed over by the speculum against the flap until the operation is completed.

After the cartilage has been removed a better view is afforded to determine if the flaps are fully free. Before closing the flaps, if the piece of cartilage left causes a tent, start in the deeper part of the nose and cut this piece toward the tip of the nose until it hangs straight, never entirely cutting it off. This heals between the flaps and gives support where it is most needed and helps prevent flapping when the nose is blown.

With Jansen-Struyken forceps cuts are made in the bony septum along the crest, also high up under the ridge; then with a pair of duckbill forceps this piece is broken out. These cuts should always be made; they not only allow the bony septum to be easily removed but, most important, prevent the possible fracture of the cribriform plate. Duckbill forceps are then used to take out the rest of the bony septum back to the sphenoid; being sure the septum is well removed between the middle turbinates, this being the most important region in the nose.

We now have the bony crest remaining. Along its upper edge is a strip of thick cartilage, the remains of the cartilaginous septum or the cartilaginous edge of a spur, which is removed with the right angle elevator.

Elevation of the membrane on the other side of the crest can now be effected by starting the right angled elevator deep in the nose and drawing it toward the tip between the crest and periosteum. The flaps being free, a V-shaped chisel is placed at the anterior edge of the crest, and with one or two sharp taps of a hammer the front and thick part is broken off. The rest of the crest is then removed, either with the chisel or the duckbill forceps.

The inside of the flaps are swabbed with 1:5,000 mercuric bichlorid to prevent infection and thoroughly dried out. The speculum is now removed and the flaps are brought together and inspected to see if they hang straight and that no ridges or spurs are left.

One suture is used in front to hold the edges of the flaps together. An Allen self-retaining speculum is inserted, the flap is held with a pair of small Hartmann forceps, and a small eye needle carrying a silk suture passed through it; then the needle is passed in the opposite direction through the membrane at the anterior edge of the cut. The suture is then tied, bringing the edges of the flaps together. This makes a figure 8 of the suture, but is the easiest tie to make. In a few days it is removed.

The outside of both flaps and the turbinates are swabbed with 1:5,000 mercuric bichlorid to prevent infection.

Plates of dental wax as wide as possible are placed on each side of the septum to support the membrane. These are left in two days to prevent the patient from blowing the nose and disturbing the flaps. No other packing is necessary.

After the wax is removed the nose is cleaned and the patient instructed to use a nasal oil two or three times a day until the swelling in the flaps is gone.

COMPLICATIONS.

Sometimes a hematoma forms between the flaps and it will be neecssary to cut the most dependent portion to let out the blood, and occasionally the piece of cartilage left will slough and an opening must be made to let out this soft material.

In all infections an injection of diphtheria antitoxin (3,000 to 5,000 units) should be given at once, as they respond quickly to this treatment. Antitoxin is used because it is easily obtained at any drug store and furnishes the foreign protein which increases the phagocytosis. In some cases a thick meaty membrane forms on the septum and turbinates; it is not diphtheria but responds to the antitoxin.

The flaps in infected cases are always thick and the patient uncomfortable because of the breathing. Steaming the nose gives the most relief.

Tears in the mucous membrane and thin spots from ulceration are smeared with an ointment of scarlet red (10 per cent); these places heal nicely under this treatment.

Soft hypertrophies on the posterior ends of the lower turbinates should be removed at this time; also any polyps coming from the ethmoids.

The middle turbinates are to be broken over to give better drainage if the patient is having sinus trouble.

If, after the operation is completed and there is a tear through both flaps, not too large a hole can be closed by the following method: A cut is made through the larger flap as high up as possible and extending as long as necessary to allow this flap to drop or fold down, covering the tear in the other flap. If the original flap is used it must be left attached in front, and must have as much membrane as possible over the tear; just barely covering it will not do, as mucous membrane flaps always retract.

Nearly all the perforation during submucous resection are due to sharp angular spurs, which occur most frequently at the upper end of the crest. If this occurs opposite the lower turbinate, and the flap method is not sufficient, the larger turbinate opposite the tear should be scarified and the septum placed over against it. A plate of dental wax is placed against the septum on the opposite side, the packing outside this; it is thus possible to change the packing every other day without disturbing the wound. In about a week it will become adherent, and as soon as the adhesion is firm a piece of the mucous membrane of the turbinate is cut out, closing the hole.

In cases of old perforations following a submucous resection, the technic is the same, except that the edges of the hole must also be scarified, and, if necessary, the turbinate can be partially detached and brought out to meet the septum.

About a month or two after the operation, if the patient is still unable to breathe easily, the soft hypertrophies on the septum are to blame. If these are deeply cauterized a roomy nose is the result as soon as the burn heals. But one side is to be cauterized at a time. If the membrane is lax and flutters when breathing, it can be tightened by cauterizing the thicker areas high up and back.

INTRANASAL EXENTERATION OF THE ETHMOIDS.

To do an exenteration of the ethmoids under the middle turbinate and conserve the turbinate, it is necessary to have a space of 5 mm. when the tissues are thoroughly contracted. If this space is not available a submucous resection of the septum should be performed. Polypi, lobuli and soft hyperplasias of the anterior ends of the middle turbinates that the snare can engage should be removed. If there is a cystic middle turbinate present the operation for the cystic middle turbinate should be performed before the exenteration.

There are two anatomic points that should be kept in mind: First, we can roughly measure to the posterior wall of the sphenoid by marking a line on the temple, half way between the temporoorbital edge and the bony auditory orifice. This point marks the posterior wall of the sphenoid. With this known point and knowing the average anteroposterior measurements of the ethmoids and sphenoid, we can by internal and external measurements during the operation easily tell just where we are in these important structures.

Second, the inner canthus of the eye is on a level with the cribriform plate and is also the position of the internal orbital plate; so this external point can be used to judge the direction when breaking into the anterior ethmoid.

The instruments necessary for the operation are a nasal speculum and a pair of nasal cutting forceps (Gruenwald, Myles, Hartmann), with a fenestrated blade 5 mm. wide, which is about one-half the width of a normal ethmoid capsule. I find the forceps with the universal handle the best, because the lower lip of the blade is stationary, and the blade can be placed just where it is wished to cut, and there is no pulling back in the action. Another instrument needed is a Pratt double end cup-shaped curet, 22 cm. long, with one end bent at an angle of 45 degrees, about 2 cm. from the end, the cup in the angle end to be fenestrated. The cup on the straight end should be 1 mm. shallower than the angle end, which gives it a sharper point to more easily open into the ethmoid when necessary.

The same anesthesia (blocking of the anterior ethmoid nerve and the sphenopalatine ganglion) is used as in the submucous resection of the septum, only blocking the side to be operated.

With the patient in a sitting position and the head thrown well back, the anterior attachment of the middle turbinate is just below the visual line to the internal canthus. The open cutting forceps is placed under the middle turbinate with the upper blade just under the anterior attachment and pressure made on a line with the internal canthus, breaking into the anterior ethmoid cells, biting backward and upward as long as there is any soft bone encountered.

As we progress backward for a complete exenteration the head is brought forward so we can follow along the plate of the skull. With a mental picture of the ethmoid capsule, bounded as it is on the orbital side by the lacrimal bone and the ethmoid orbital plate, nasally by the middle turbinate, superiorly by the temporoorbital plate and posteriorly by three-fifths of the anterior wall of the sphenoidal sinus, we take the straight end of the curet and with a firm but gentle stroke in every direction curet out all the soft tissue. If we find firm, smooth and yielding tissue under our curet on the orbital side we know we are down to the periosteum of the orbit and force should not be used.

The anterior cells are removed with the angle end of the curet, curetting in an arc form toward the tip of the nose, thus removing the anterior cells and enlarging the space upward toward the frontal sinus. We now cut out the floor of the capsule back to the sphenoid sinus, giving a free drainage under the entire length of the middle turbinate. There is little bleeding, owing to the fact that we do not cut either the anterior or posterior ethmoid or the sphenopalatine arteries.

The cavity is wiped out with large swabs, using a whirling movement to collect any particles of bone and débris, and then a large swab of cotton saturated with an iodin and glycerin solution is applied in the cavity for a few minutes to stop the bleeding and disinfect it. With a dry cavity any overlooked soft portions can be removed.

The cavity under the turbinate is not packed, but a finger-shaped piece of cotton is placed in the middle meatus back to the anterior end of the middle turbinate, and the patient is allowed to go home, with instructions to remove the cotton in four hours and report in three days. The home treatment consists of dropping a few drops of nasal oil into the nostrils three times a day, the patient in the supine position with the head back; as the medicine is dropped into the nostril, he is told to sniff violently. Every third day, after cocainizing and cleaning the nose, the cavity under the middle turbinate is swabbed with the iodin-glycerin solution. It will frequently be found necessary to bite away granulations to keep the cavity clear and healing properly. In a short time it is hard to detect that the nose has been operated upon.

If, after a suitable length of time, the discharge does not cease, the nose can be recocainized and the operation easily extended into the frontal, sphenoid or any adjacent cells that may have been overlooked.

INTRANASAL FRONTAL SINUS OPERATION.

We are becoming more convinced that it is seldom necessary to do a radical frontal sinus operation or an intraexternal operation like the Lothrop.

Unless the symptoms show a brain involvement, we believe that, if the natural ostium is enlarged enough to give free drainage, and the discharge removed by suction if necessary, this treatment will show the greatest percentage of recoveries. It is extremely simple after the anterior ethmoids have been exenterated by the "under the middle turbinate method" to enlarge the frontal sinus opening with the rasps, as recommended by Dr. Good years ago. The lateral space available for an intranasal opening into the frontal sinus is from 5 to 7 mm. This is a little less than the width of the articulation of the ethmoid bone with the frontal. A workable outline shown on the patient is the space between the internal canthus and the central perpendicular line of the nose.

OPERATION.

Instruments: Nasal speculum, Gruenwald biting forceps No. 3, Pratt double end ethmoid curet, frontal applicators, Good-Thompson rasp set.

Anesthesia: Blocking of the anterior ethmoid nerve and sphenopalatine ganglion, following the ethmoid exenteration with applications of cocain on curved applicators to the frontal sinus duct region.

The cutting forceps is placed open just under the anterior attachment of the middle turbinate. With the tip of the movable blade pointing toward the internal canthus, pressure in this direction will break into the anterior ethmoid cells.

The cells are bitten away to the frontal plate and backward as far as the posterior wall of the anterior ethmoid, which is recognized in nearly every case by the firmer partition. This space is smoothed by the straight end of the curet. The angled end of the curet is now inserted with the cup pointed forward, and curetting motions are made in an arc form toward the tip of the nose. The smooth, firm bone will be recognized as soon as all cells have been broken down and removed. Bleeding can be stopped, if necessary, by swabs dipped in the iodin-glycerin solution, or epinephrin chlorid. With beginners it is well to attempt passage of the frontal probe of the rasp set, and if successful the little groove on the rasp placed on the probe gives the correct direction for the point of the rasp. After a few trials the rasp is used directly without the use of the probe.

To find the frontal opening, take the sharper curved rasp and pass it under the attachment of the middle turbinate into the exenterated space of the anterior ethmoid. Point the tip of the instrument toward the frontal sinus in an imaginary space marked by a central perpendicular line with a parallel line passing through the internal canthus. By working delicately upward with a rasp motion in this direction you will either find the normal opening or break through intervening cells into the frontal sinus. The rasp cuts on the pull, so the opening is enlarged accordingly. The opening should be large enough to pass with ease a 3 mm. cannula. This size is maintained by using frontal dilators until the case is cured. If granulations form or the hole contracts, the rasp is used again.

OPERATION ON THE CYSTIC MIDDLE TURBINATE.

The nasal speculum, cutting forceps and snare are the instruments usually needed in this operation.

The cystic middle turbinate generally has a lobular deformity, which when snared off opens the cyst. If there is no deformity, but simply a large turbinate, the cutting forceps will, as a rule, easily break into the cyst; but should it be too dense some sharper instrument, such as a chisel, will make a point of entry for the cutting forceps. The point of entry should be in the lateral half of the anterior end. It will be seen on opening that the lateral wall of the cyst is extremely thin and should be bitten away, leaving the heavier mesial wall to become the future turbinate. As the cyst is lined by mucous membrane, the turbinate is soon well and completely covered. It is surprising how soon it will thicken and return to form.

If the ethmoid is diseased the exenteration of the ethmoid is continued at this point; otherwise the turbinate is broken toward the lateral wall and packed in this position. The packing is removed in two days and reapplied if necessary.

ANTRUM OPERATION.

A semiradical antrum operation easily performed is as follows: The parts are anesthetized by blocking the anterior ethmoid nerve and the sphenopalatine ganglion as in the submucous operation and placing a swab under the lower turbinate. It is best to wait at least twenty minutes.

The anterior attachment of the lower turbinate is cut far enough to allow the turbinate to be displaced upward, giving a good view of the lateral wall underneath.

With the small curved chisel an opening is made into the antrum about one-half inch posterior to the anterior attachment of the lower turbinate, and the chisel turned to make a round opening. The Good-Thompson frontal rasp is now used and the lateral wall anterior to the chisel hole is rasped away, exposing the anterior angle of the antrum. The Gruenwald cutting forceps are now adjusted so as to give a horizontal cut. They are introduced closed into the nostril and, when allowed to open, the active blade will slip into the opening made by the rasp and the lateral wall posteriorly is removed as far back as necessary.

The antrum can now be curetted by angular curets and allowed to remain open. The turbinate is now replaced. The biting forceps can be used at any time to remove granulations to keep the opening free.

HYPERTROPHIES AND HYPERPLASIAS.

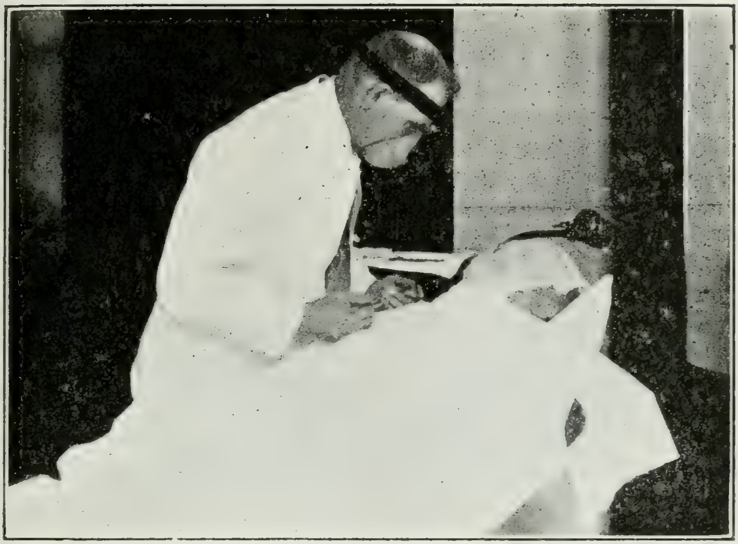
We find in our work that simple hypertrophies of the nose, when caused by a deflected septum or sinusitis, will gradually recede when proper ventilation is restored and the sinusitis cured. When this hypertrophy has progressed to hyperplasia we find it necessary to remove certain portions until the turbinates conform to the normal.

We believe that this work should not be hurried or too much removed at one sitting. The rapidity of covering the denuded portion with healthy mucous membrane is hastened by limiting the amount of destruction. It is necessary to always keep in mind the relative size of the normal structure that occupies the space, to avoid removing any portion necessary to its proper restoration.

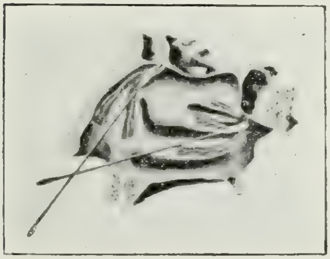
The soft, irregular hyperplasias, whether situated on the anterior or posterior ends or hanging from any other portion of the turbinates, can be easily removed with the snare.

By using the snare we are not removing any undue amount of tissue. When the snare cannot be used the biting forceps can be employed under inspection and the turbinate trimmed to normal contour.

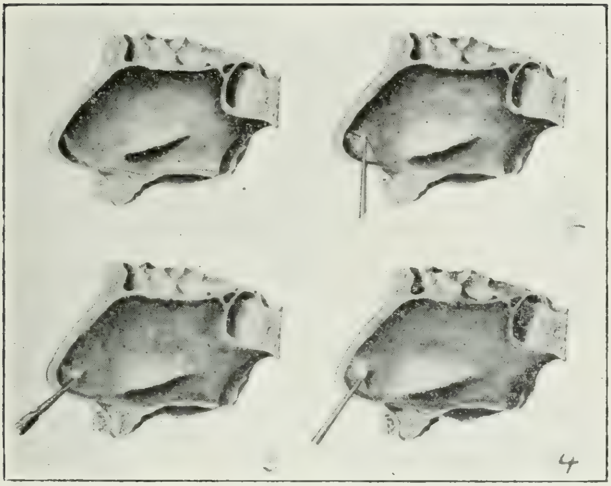
919 METROPOLITAN BANK BUILDING.



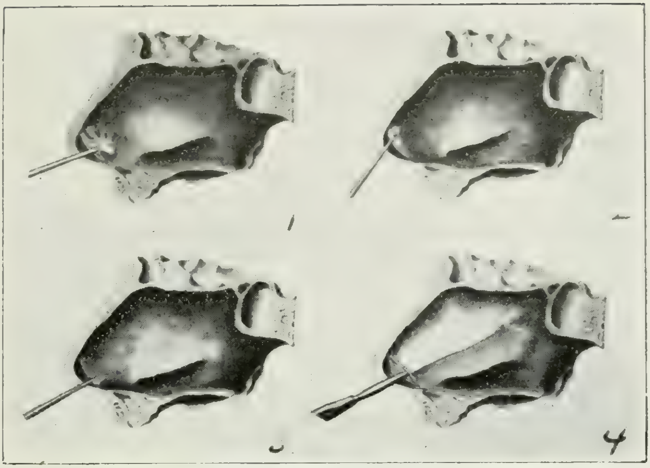
Position of patient during operation.



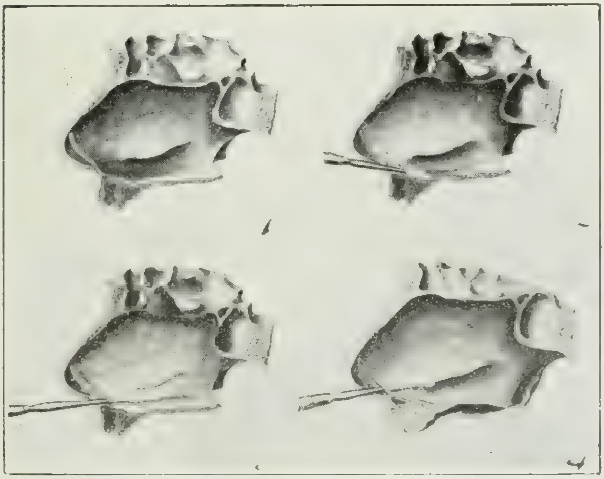
Cotton applicators blocking anterior ethmoidal nerve and nerves from spheno-palatatine ganglion.



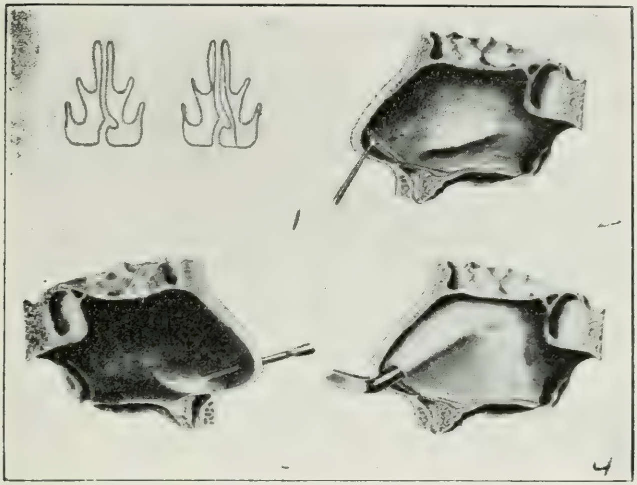
1. Cut through mucous membrane and perichondrium.
2. Pushing membrane back over the cartilage.



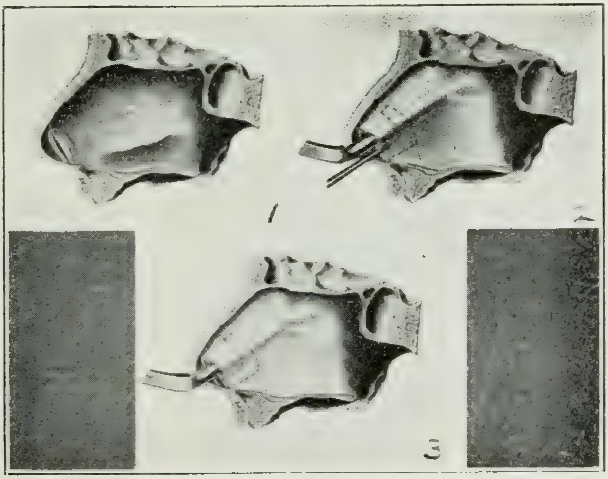
1. Elevating lower part of flap near cut from the inside.
2. Completing original cut from inside out at upper end.
3. Completing original cut from inside out at lower end.
4. Elevating flap above spur.



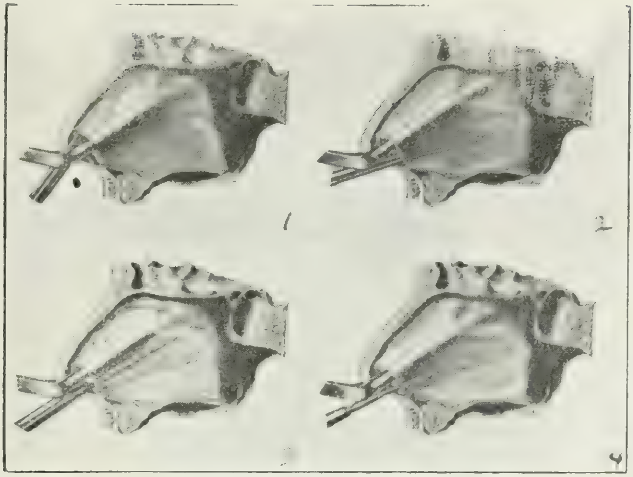
1. Completing original cut down on the floor.
2. Elevating membrane on the floor.
3. Elevating membrane on the under side of spur.
4. Elevating membrane on the edge of spur.



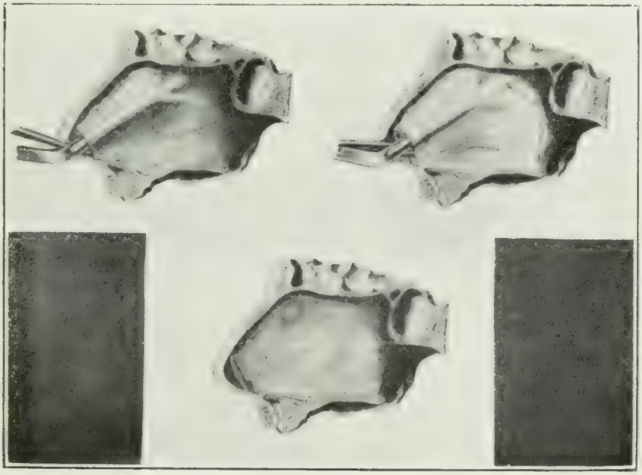
1. Shows flap elevated on one side out on floor.
2. Cut through the cartilage.
3. Elevating the membrane on the opposite side well down on the floor in front.
4. Speculum holding flaps apart.



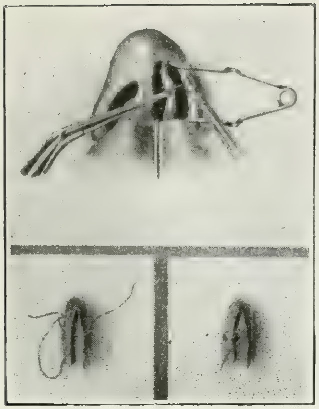
1. Small slice taken off cartilage at the original cut.
2. Piece of cartilage taken out by Ballenger knife.
3. Cut at posterior edge of cartilage.



1. Struycken's forceps making cuts in bone below.
2. Struycken's forceps making cuts in bone above.
3. Bone being removed by duck bill forceps.
4. Remains of cartilage along the crest removed.



1. Anterior part of crest broken by chisel.
2. The remainder of crest removed by duck bill forceps.
3. Shows amount of cartilage and bone removed.



Method of placing suture.

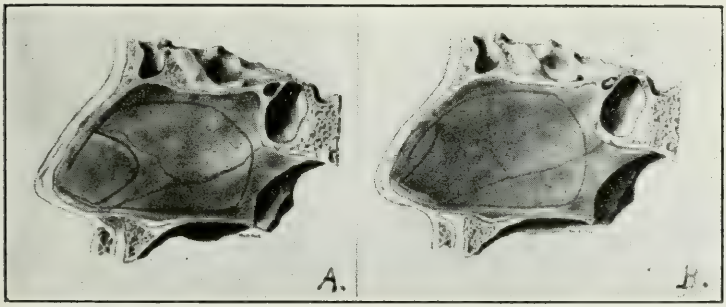


Fig. 13.—Submucous operation: A, largest piece of cartilage left, also cut if necessary to make it hang straight. B, smallest piece of cartilage and cut if necessary.

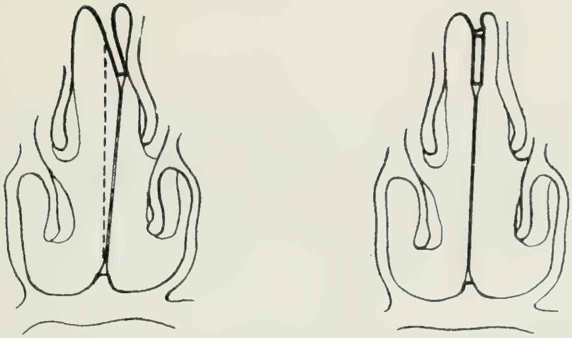


Fig. 14.—Submucous operation: A, tenting of membrane if bent cartilage is left; B, cut in cartilage to make it hang straight.

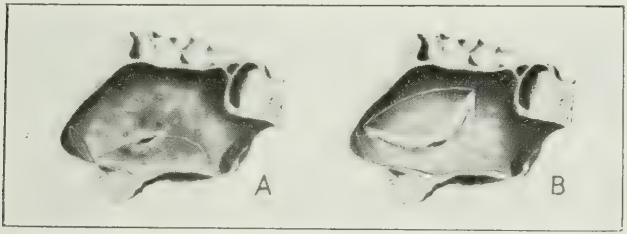


Fig. 10.—Submucous operation: A, flap with tear on one side, perforation in the other; B, sliding flap to close perforation.

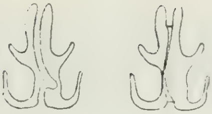


Fig. 11.—Submucous operation: Using lower turbinate to close perforation.

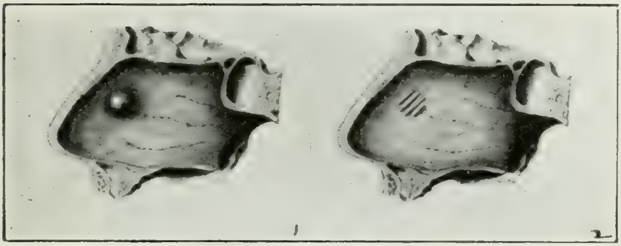


Fig. 12.—Submucous operation: A, soft hypertrophy on septum; B, cautery marks in soft hypertrophy.

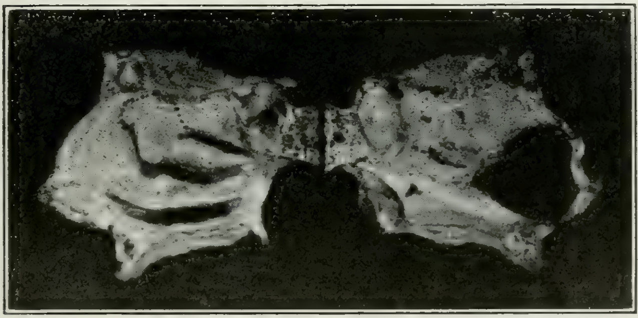
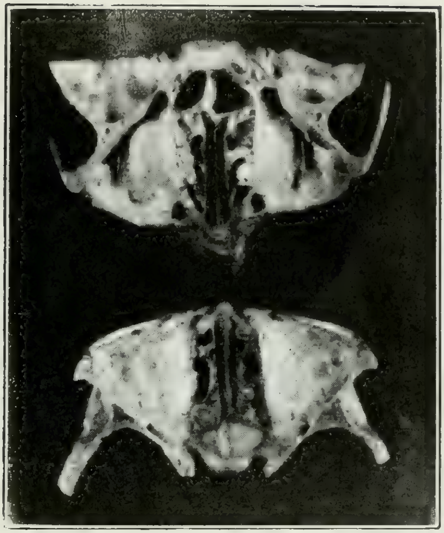


Fig. 1.—Ethmoid operation: Lateral wall of nasal cavity. The attachment of the so-called middle turbinate is outlined. The anterior ethmoid cells are covered by the anterior half of the middle turbinate. The posterior ethmoid cells are covered by the so-called superior turbinate.



A horizontal cut through the ethmoid capsule just below the cribriform plate, showing the bony walls of the capsule, and how the ethmod cells and sphenoids follow each in sequence anterio-posteriorly.

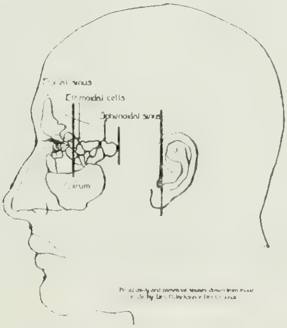


Fig. A—Ethmoid operation: Lateral view of the head, showing the relative position of the paranasal sinuses. Three perpendicular lines mark the orbit edge, the bony auditory canal and the posterior wall of the sphenoid sinus.

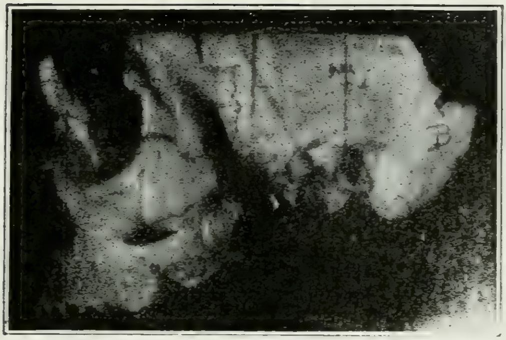


Fig. B.—The three points marked by perpendicular lines shown on the skull.

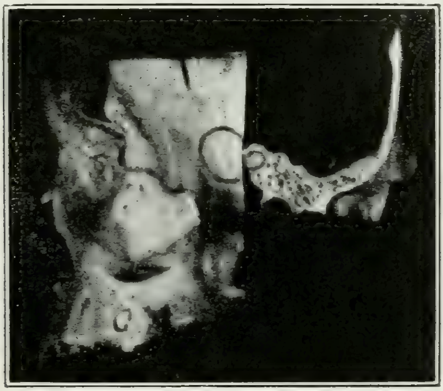


Fig. C.—The skull cut through at the middle perpendicular line, showing that it marks the posterior wall of the sphenoid sinus.

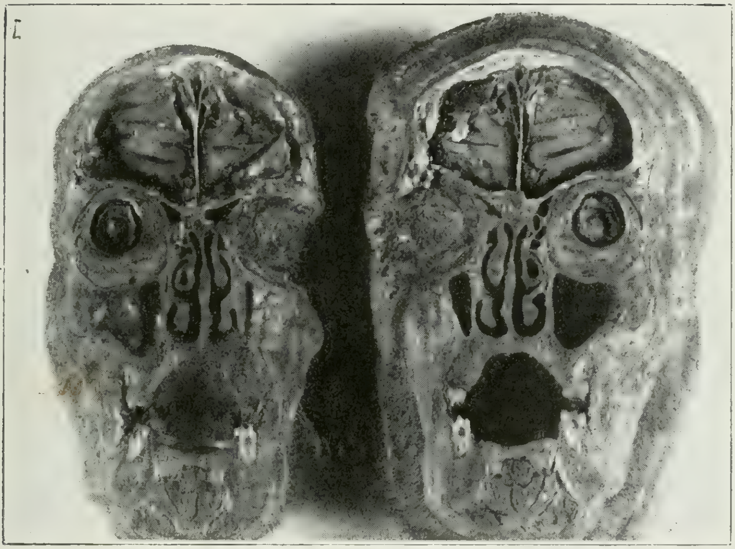


Fig. 1.—Transverse section of the head just posterior to the anterior attachment of the middle turbinate.

A. Showing the over hang of the turbinates.

B. Cystic condition of the middle turbinate.

C. In going up under the anterior end of the middle turbinate the anterior ethmoid cells are immediately entered.

D. How the nose portion of the floor of the frontal sinus is formed by the ethmoid cells.

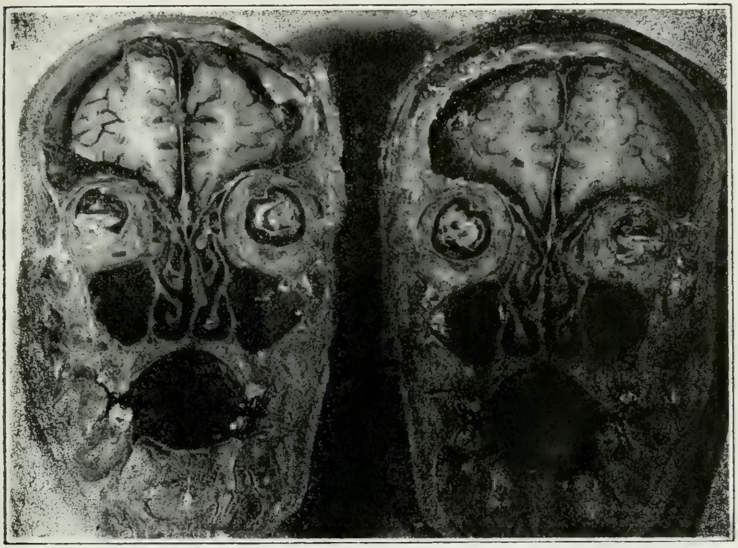


Fig. 2.—Transverse section of the head, one c.m. posterior to the previous section.

A. The bony walls of the ethmoid capsule.

B. The cribriform plate on a level with the center of the eye balls.

C. Infundibulum.

D. Antro-ethmoidal cell.

E. The thin partition between the antrum and lower meatus, and the ease of operating at this point without destroying any part of the lower turbinate.

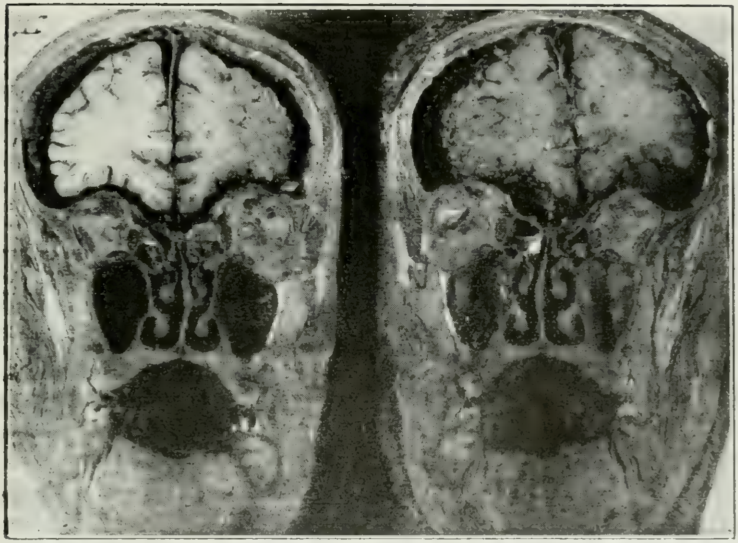


Fig. 3.—Transverse section of the head, one c.m. posterior to section II.

A. The continued bony walls of the ethmoid capsule.

B. The anterior walls of the posterior ethmoids.

C. The greater size of the posterior ethmoids.

D. How the posterior ethmoids are directly posterior to the anterior ethmoids.

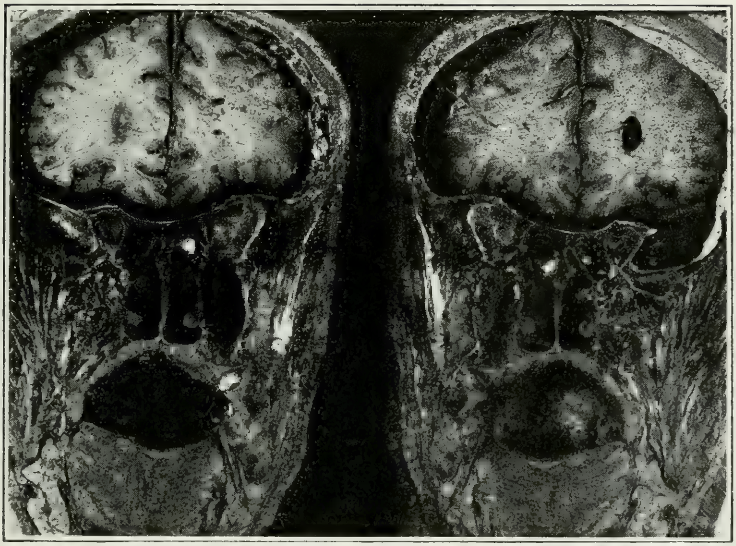


Fig. 4.—Transverse section of the head, one c.m. posterior to section III.

A. The posterior wall of the posterior ethmoid cells.

B. The ostia of the sphenoids.

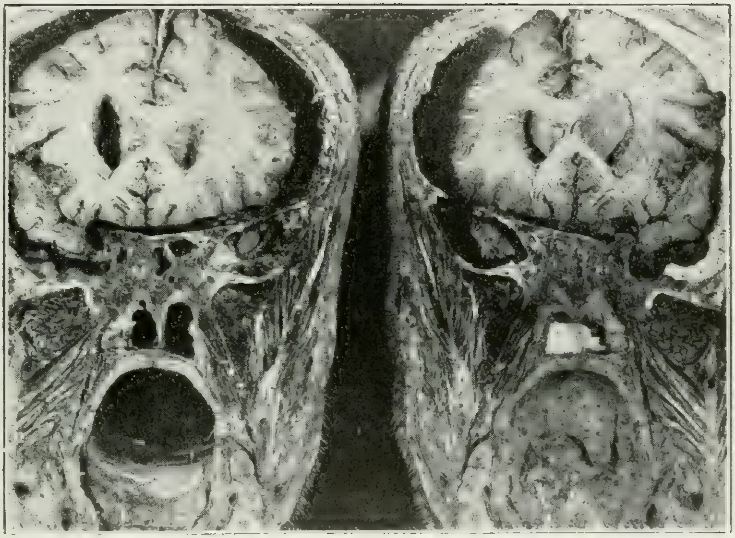


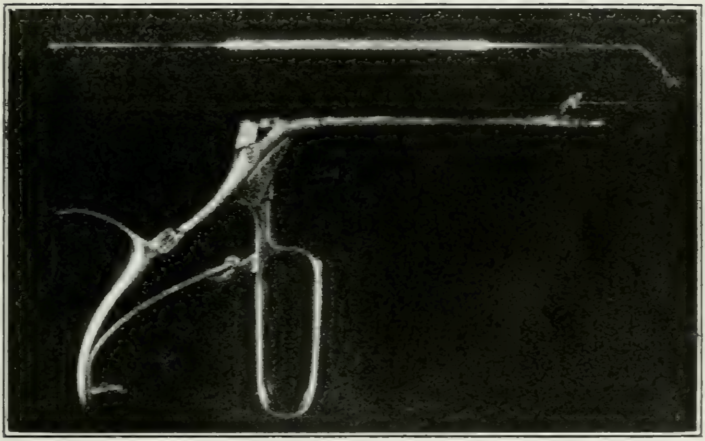
Fig. 5.—Transverse section of the head. one c.m. posterior to section IV.

A. Posterior wall of the sphenoids.

B. The large posterior ethmoid cells on the right in direct contact with the right optic nerve.

C. The membrane covering the posterior wall of the left sphenoid sinus is intact.

D. The small size of the sphenoid sinuses.



Ethmoid Operation.

1. The Pratt ethmoid curet.
2. The Gruenwald cutting forceps.

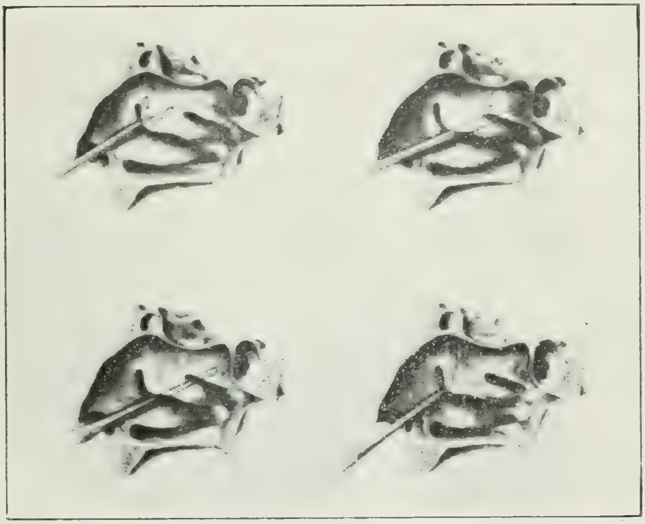


Fig. 3.—Ethmoid operation: A, Gruenwald forceps biting out the anterior ethmoid cells under the middle turbinate; B, the biting continued backward into the posterior ethmoid cells; as the partition walls of the cells are broken, the mesial wall of the capsule springs toward the septum, giving a large working space; C, curetting the cavity smooth with the straight end of the curet; D, removing the anterior and infundibular cells from the nasal floor of the frontal sinus.

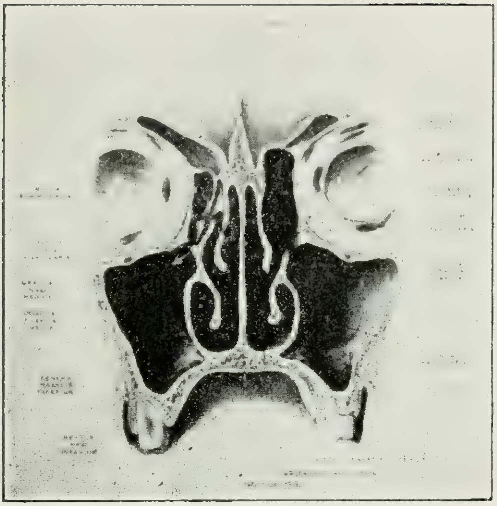


Fig. 4.—Ethmoid operation: Exenterated ethmoid region. The protection afforded by leaving the middle turbinate from injuring olfactory fissure or cribiform plate. The bony wall outlining the ethmoid capsule. This illustration is modified from a cut in Loeb's Operative Surgery of the Nose, Throat and Ear.

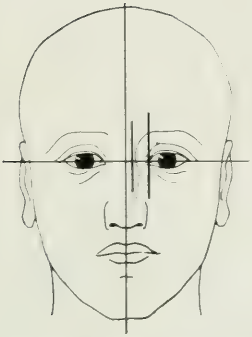


Fig. 5.—Intranasal frontal sinus operation: The central perpendicular line marks the septum. The horizontal line marks the position of the cribiform plate. Between the heavy and light perpendicular lines lies the olfactory fissure. Between the heavy perpendicular lines lies the ethmoid capsule; direction the instrument is to take to enter the frontal sinus intranasally.

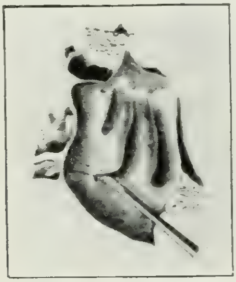


Fig. 6.—Intranasal frontal sinus operation: Rasp under the middle turbinate into the frontal sinus after exenteration of the anterior ethmoids.

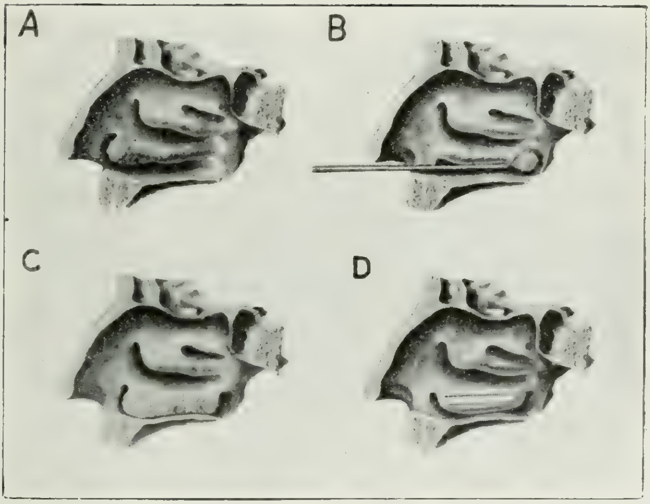
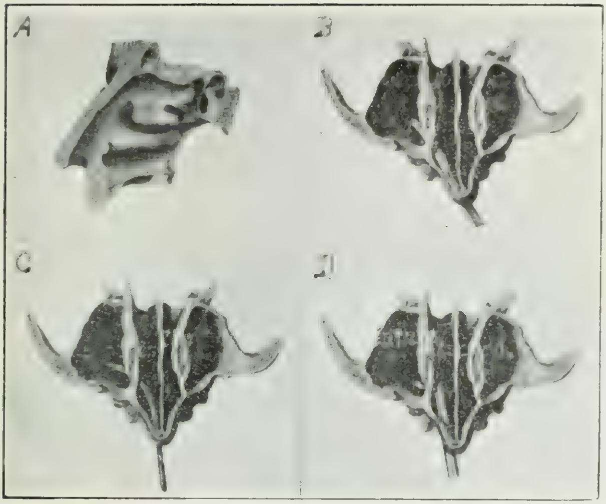


Fig. 7.—Operation for hyperplasias: A, hyperplasia of anterior end of lower turbinate removed by snare; B, removal of the hypertrophied posterior end of the lower turbinate with the snare; C, hyperplastic lower turbinate reduced by gradual snaring and cure of sinus diseases; D, hypertrophied lower turbinate reduced by cautery.



A. A three-fourths view of the lateral wall showing the anterior attachment of the lower turbinate detached and the turbinate bent upward, exposing the antrum wall.

B. The antrum wall under the lower turbinate punctured by the chisel.

C. The rasp in position to remove the anterior portion of the wall.

D. The biting forceps in position to bite away the wall posteriorly as far as desired.

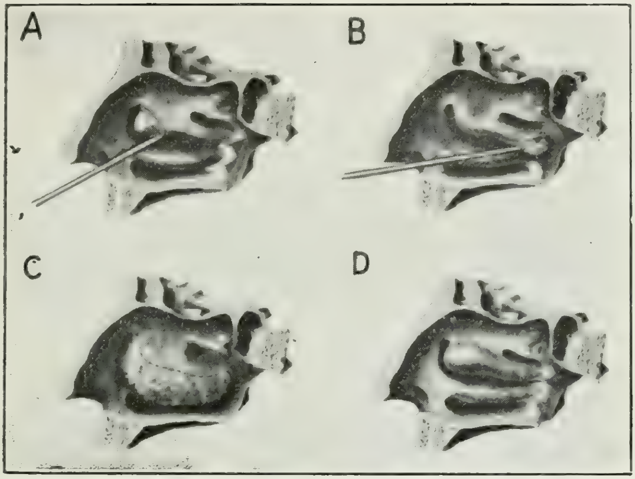


Fig. 8.—Operations for lobulus and hyperplasias: A, removal of lobulus with snare; B, removal of posterior hyperplasia of middle turbinate with snare; C, hyperplasia of middle turbinate complicated by hyperplastic ethmoiditis and adhesions to septum; reduced to normal by exenteration of ethmoid cells and gradually snaring away the hyperplastic tissue; D, cystic middle turbinate reduced as in Figure 9.

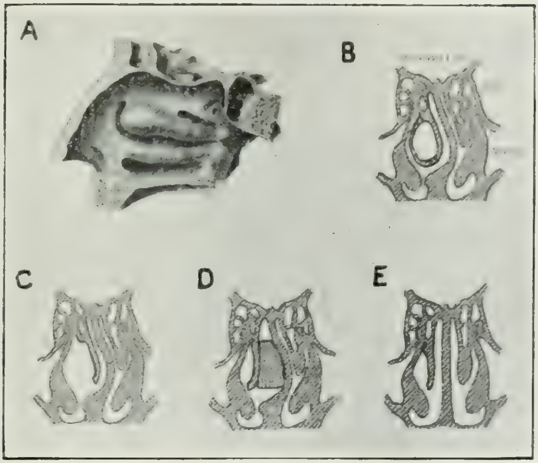


Fig. 9.—Cystic middle turbinate operation: A, cystic middle turbinate; B, transverse section showing cyst exposed and points where biting forceps start to remove the lateral wall; C, lateral wall removed; D, remaining portion of turbinate broken over and held in place by Simpson splint; E, submucous resection performed to remove septal deformity.

III.

GRAVITY DRAINAGE AND SWABS VS. IRRIGATION AND WICKS IN OTITIS MEDIA.

By George E. Barnes, B. A., M. D.,
Herkimer, N. Y.

Acute suppuration in the recesses of the middle ear, especially in patients with little resistance on account of bad general health, certainly presents difficult problems in treatment.

Irrigations wash pus out of the external auditory meatus and, through the incision in the membrana tympani, to some extent out of the middle ear, but the current of fluid certainly carries infective material from the meatus into the middle ear. Such a procedure cannot be said to be based on good surgical principle, and solutions of strong antiseptics cannot be used in the ear. Indeed, the avoidance of superinfection and reinfection of the middle ear is one of the cardinal points which should be observed in treating middle ear disease. Such added infection takes place with remarkable facility. It is a wonder that suppuration ever disappears under treatment by irrigations. Furthermore, as is well known, irrigations alone often fail to dislodge all the purulent accumulation in the meatus.

Does the gauze wick placed in contact with the incision in the membrane drain the pus out of the middle ear and along the meatus in an acceptable manner? Certainly not. It is well known in general surgery that gauze drains in the presence of pus do not drain but act as plugs. They very quickly lose their capillary action because the mashes become filled with pus, which, unlike thinner fluids, as volatile oils and water, is too thick to flow along gauze. This is especially true when air has access to the wick and dries the pus in the meshes. To be sure, this drying of pus in the meatal wick is considerably prevented by the wad of cotton in the concha which cuts off repeated access of air.

Now, if the prevailing treatment of otitis media by irrigations or wicks or the combined use of both is not based on good surgical principles, is it possible to improve on these methods? Yes, by making use of gravity drainage, absorbent pads in the concha, and meatal cleansing by means of swabs, while the lips of the incision are kept from adhering by use of my spatula.

For good gravity drainage the patient should lie about half or more of the time with the affected ear on the pillow (which should be covered with a towel to catch any little pus that may leak out). Nearly all pus is carried outward along the meatus and is taken up by a rather loose wad of absorbent cotton placed in and filling the concha. This pad of cotton should be changed whenever it becomes saturated on its inner surface, and this is not much oftener than irrigations are practiced by those who believe in them. The concha should be washed with soap and water once or twice daily.

Every day or every other day the doctor should very gently swab out the meatus with a cotton wrapped applicator. This little procedure should be done with the utmost care. The applicator should be made of copper wire of about .03 inch diameter and of just sufficient hardness to prevent twisting while the cotton is being rolled on it. Such an applicator has sufficient resilient flexibility so that even when a trifle more pressure is made with it than intended it will bend easily and thus not cause, when correctly dressed with cotton, pain to the patient or injury to the tissues. I could not find such applicators on the market and had some made to order. (The ends of these applicators should be triangular in cross section so as to hold the cotton as the applicator is rotated.) Before using, the end of the applicator should be slightly bent to correspond with the curve of the inferoanterior wall of the meatus so that pus can be easily gathered up from the deepest recesses.

The flat pledgets of cotton which are to be rolled on the applicator should be carefully prepared. Of course, the hands should be thoroughly cleaned before the cotton is touched, and during the whole seance nothing should contaminate the ends of the thumb and index finger used in rolling the pledgets on the applicator. A thin layer is peeled off from a small handful of cotton, and from it are pulled flat pledgets of a size found to make a swab of just the right size to pass readily through the meatus without friction and to gather up the maximum quantity of pus. The pledget should not be too thin on the end that is to form the business part of the swab, and if thus thin when first pulled off it can be folded back a little. The pledget should be rolled on the applicator only so tightly as to prevent it from coming off during use and to permit ready removal after use. It should be rolled quite tightly about the extremity of the applicator, but the cotton beyond the extremity should form a spongy tuft. This preparation of the swab protects the end of the applicator from injuring the tissues and hurting the patient and furnishes a good absorbent body.

A good method of procedure is first to remove with tissue forceps the pad of cotton in the concha and cleanse the concha; next to cleanse the outer half of the meatus with dry swabs and then with a swab wet with a strong solution of boric acid; and lastly, to cleanse the inner half of the meatus in the same manner, the whole meatus being finally swabbed with boric acid solution and partially dried. Care should be exercised not to make any or but slight pressure against the membrane.

If on inspection the edges of the incision in the membrane are found to be adherent in places and the membrane is still bulging and pulsating considerably, my spatula is gently inserted merely the depth of the incision and passed from one end to the other, avoiding any unnecessary spreading of the lips to prevent the entrance of fresh infection and disturbance of anatomic parts. The use of the spatula promotes drainage and prevents the closure of the incision except necessarily as it grows up at the ends. (Of course, the spatula may be used with the irrigation treatment if ever needed, but usually the force of the stream is apt to open the incision even too widely.)

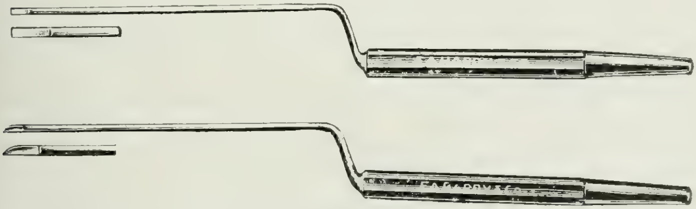


Fig. 1.

IV.

TONSILLAR TUBERCLES CONTAINING INTRACELLULAR CONCRETIONS SIMULATING FOREIGN BODY PSEUDOTUBERCLES.*

By Carl Vernon Weller, M. S., M. D.,

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Ann Arbor.

In contrast to Sewall,[1] who did not find a single "foreign body giant cell" in the examination of 1,544 tonsils, we have found such giant cells to be of frequent occurrence. Either with or without accompanying epithelioid or fibroblastic proliferation, they constitute foreign body pseudotubercles, which, it has been stated by some, resemble true tubercles so closely that the diagnosis of tonsillar tuberculosis by histologic methods alone is thereby somewhat invalidated. We do not consider this objection to be well founded, for the foreign body pseudotubercles are as a rule readily differentiated by the presence of the foreign body itself or of a vacuole which represents its former location, provided it has been soluble in the reagents used in the preparation of the section. Attention to this detail and to the more irregularly syncytial character of the giant cells enveloping gross foreign bodies should prevent the error of mistaking the foreign body pseudotubercle for a true tubercle. To call attention to the fact that it is possible for the opposite error to occur is the purpose of this paper. If, through necrosis and subsequent calcification of the interior, a giant cell comes to exhibit an intracellular concretion, such a true tubercle may erroneously be considered a foreign body pseudotubercle. We have found such intracellular bodies in several cases of undoubted tonsillar tuberculosis.

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A prolonged search of the literature has provided but two descriptions* of tonsillar tuberculosis showing intracellular concentric concretions of the sort here described. One of these authors considered the possibility of confusion with foreign body pseudotubercles. Gottstein[2] (1896), in describing the microscopic appearance of his case IV, writes:

"There are typical Langhans' giant cells in the tubercles. Within some of the tubercles peculiar bodies are found. These are observed only in a few sections and in but two areas near a connective tissue septum. In a large giant cell there is to be seen a concentrically laminated body, staining intensely with hematoxylin and having a clearer center in which there is a peculiar stroma or network. In some cases this body appears to extend beyond the giant cell. These bodies give the impression of being composed of lime salts, which is confirmed by examination with Nicol's prisms." After considering the possibility of laminated islands of epithelium becoming calcified and producing foreign body pseudotubercles he concludes that this case must be considered a combination of true tuberculosis with foreign body pseudotuberculosis, lacking, however, absolute proof of either, since neither tubercle bacilli nor cellular inclusions could be demonstrated. It is entirely evident from his discussion of the case that he felt that no positive diagnosis of these peculiar calcareous bodies could be made.

Uffenorde[3] (1903) was aware of this observation and refers to it in his description of the microscopic findings in his own case III. In an area of numerous epithelioid tubercles with many very large giant cells, he found one giant cell in which there was a concentrically laminated structure with peculiar border and clefts which he recognized as being like those described by Gottstein. He does not, however, discuss its significance.

In three instances, among 142 cases of microscopically verified tonsillar tuberculosis from 8,600 pairs of tonsils examined,[4] we have found concentrically laminated concretions of the type noted by Gottstein and Uffenorde. These may be presented briefly as follows:

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*Grosvenor (Journ.-Lancet, Minneap., 1913. XXXIII, 653-656), figures these concretions very characteristically and recognizes their calcareous nature in the legend, but does not refer to them in his text.

Case No. 2659-U. Male, age 17. Bilateral tonsillectomy for so-called septic tonsils. Microscopic diagnosis: One tonsil shows only a chronic hyperplastic tonsillitis with hyperkeratosis and increased stroma. The other tonsil shows numerous tubercles scattered throughout about two-thirds of its substance. These are chiefly epithelioid in type, some with central caseation. Giant cells are numerous and large, and in the interior of many of these there are concentrically laminated bodies, staining deep bluish purple with hemalum and eosin, apparently calcareous in nature. Advanced unilateral tonsillar tuberculosis.

Case No. 501-V. Machinist, age 24. Double tonsillectomy for recurring tonsillitis. Microscopic diagnosis: Chronic hyperplastic tonsillitis. Large colonies of cocci. Hyperkeratosis. Bone and cartilage formation in the capsule. In each tonsil there are groups of epithelioid and giant cell tubercles localized about a single crypt. In one tonsil these are chiefly epithelioid and tend to become confluent without caseation. In the other, tubercles are much more numerous and contain giant cells, in many of which there are small blue staining (with hemalum and eosin) bodies. Some of these concretions have a more lightly staining area in the center and practically all show some degree of concentric lamination.

Case No. 1273-W. Female, age 10. Comes from an institution for dependent and defective children for bilateral tonsillectomy. No further history. Microscopic diagnosis: Both tonsils show a moderate chronic hyperplastic tonsillitis and in both there are large numbers of scattered epithelioid tubercles, many of which show an active caseation. In both tonsils there are, within undoubted tubercles, giant cells that contain laminated bodies, apparently concretions. The smallest of these bodies stain a light blue with hemalum and eosin, while the larger ones, some of which occupy nearly the entire interior of a giant cell, stain a deep purplish blue. Active bilateral miliary tonsillar tuberculosis.

The bodies thus noted as occurring within the giant cells of true tuberculosis vary in size from minute bluish dots up to masses almost filling the largest cells. They are usually rounded in outline, although rarely perfectly spherical, and in most cases show a definite concentric striation or lamination which is much more evident upon focusing upon different levels than it can be made to appear in photographs. Their density greater than that of the surrounding cell substance, is shown by their tendency to tear out or to fragment during sectioning. The staining reactions are those of lime salts; bluish purple with hemalum, yellowish brown to brownish black by the von Kossa silver method and blue with toluidine blue. Their formation apparently results from a necrosis of the cytoplasm of the central portion of the giant cell with subsequent deposit of lime salts in or about the necrotic focus. In this way the lighter center and the concentric lamination can both be explained.

In each of the three cases noted above, other tubercles, presenting the criteria necessary for the histologic diagnosis of tuberculosis, but without the intracellular concretions, were present in the same areas with those containing them, and differing in but this one respect from them. There was no hesitancy in making a diagnosis of true tubercles, with giant cells containing concentric concretions, in each instance.

CONCLUSIONS.

1. In three out of 142 cases of tonsillar tuberculosis, diagnosed histologically, concentrically laminated concretions were found in the central cytoplasm of some of the Langhans' giant cells.

2. The presence of such concretions gives rise to the possibility of interpreting the true tubercles containing them as foreign body pseudotubercles, thus reversing the type of error usually considered in connection with pseudotubercles in general.

3. The characteristic concentrically laminated structure differentiates these concretions from the foreign bodies usually found in tonsillar pseudotubercles.

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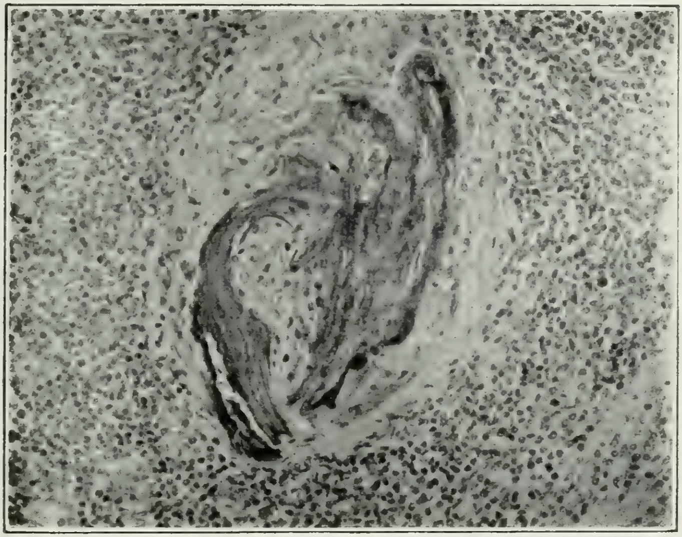

Figure 1.

Figure 1. Photomicrograph of a tonsillar foreign body pseudotubercle containing calcareous material arranged in irregular fibrillar laminated masses. Probably calcified kerato-hyalin acting as foreign body. Irregular syncytial giant cells about the foreign body. Hemalum and eosin stain. Zeiss "B" objective without ocular.

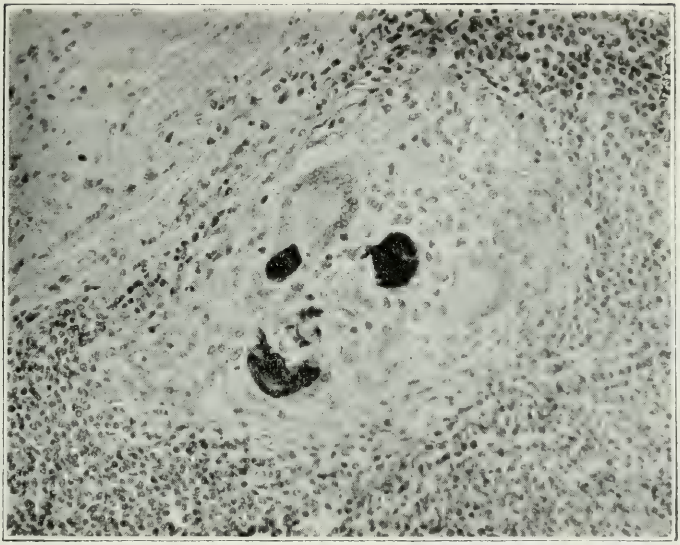

Figure 2.

Figure 2. Photomicrograph of true tubercle containing numerous giant cells and three intracellular concretions, somewhat laminated, the largest of which is, in part, torn out in the cutting. Case 501-V. Many other tubercles without concretions were found nearby in the same section. Hemalum and eosin stain. Zeiss "B" objective without ocular.

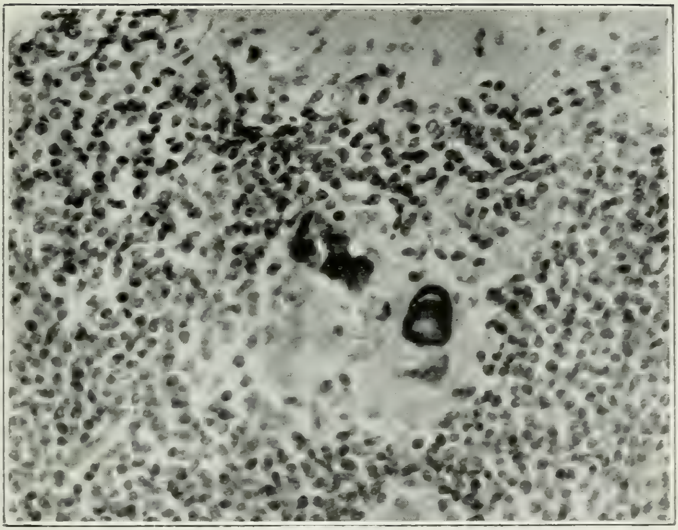

Figure 3.

Figure 3. Photomicrograph of another tubercle in Case 501-V showing concentrically and irregularly laminated concretions within and nearly replacing the giant cells. Hemalum and eosin stain. Zeiss "DD" objective without ocular.

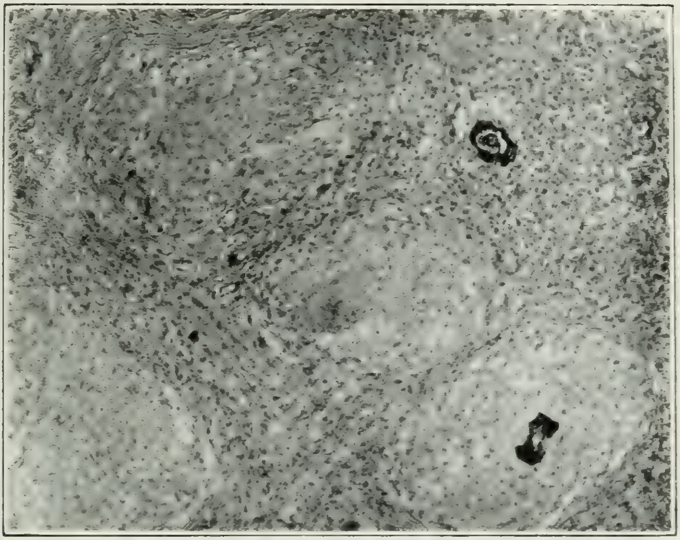

Figure 4.

Figure 4. Low power photomicrograph, Case 2659-U. Numerous tonsillar tubercles in two of which concretions occur within giant cells. One of these concretions shows very well the concentric lamination so frequently found. Hemalum and eosin stain. Zeiss "B" objective without ocular.

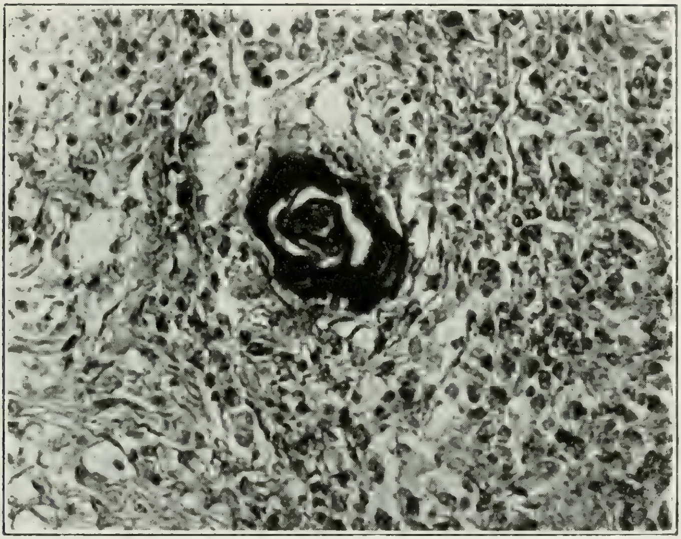

Figure 5.

Figure 5. High power photomicrograph of one of the concretions shown in previous figure. Concentric lamination and lighter staining in central portion are evident. In this, as in all other cases, lamination is much more evident while focusing up and down through the thickness of the concretion than it can be made to appear by a photograph at any one focal plane. Hemalum and eosin stain. Zeiss "B" objective with ocular.

V.

A DEMONSTRATION OF THE PHYSICS OF SUCTION APPLIED TO THE NOSE WITH MODIFICATION OF THE USUAL TECHNIC.

By Samuel Iglauer, M. D.,

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In recent years the use of suction apparatus has become very popular in the diagnosis and treatment of exudative diseases of the paranasal sinuses. The simple rubber bulb suction outfit originally used has in a large measure been supplanted by the electric vacuum pump, which is much more effective in action. Unfortunately, despite these technical improvements, but little attention has been paid to the physical principles underlying the use of negative pressure, and judging from the report of a discussion of this subject (Laryngoscope[1]), considerable confusion seems to exist regarding these principles. A disregard of the physics involved has also at times led to extravagant claims for this method of treatment, which are not warranted by the facts.

Many years ago Uffenorde[2] presented a critique of this subject and, employing a rubber suction bulb, performed some experiments on the antrum of the cadaver to explain the physics involved. Heermann[3] also explained the physics somewhat empirically and based his statements chiefly on clinical evidence.

The experiments which I have performed have been with the electric pump and are similar to but not identical with those of Uffenorde. From the standpoint of physics and for purposes of demonstration, any accessory sinus of the nose may be likened to a glass retort having a single narrow outlet. The position of this outlet varies according to the anatomic structure of the particular sinus. In the frontal sinus the outlet is at the bottom of the cavity; in the antrum and sphenoid sinuses it is very near the top; with the ethmoids the position of the outlet varies with each individual cell.

If such a retort be partially filled with water and have its neck connected by tubing with a suction pump, and if in addition a vacuum gauge be attached, air tight, above the fluid in the retort, we can reproduce very closely the physics of artificial suction applied to the paranasal sinuses.

Experiment I. With the retort connected as described above, with the level of the contained fluid below the outlet of the retort, it is evident that upon creating a partial vacuum in the outlet of the retort there will be no movement of the fluid within the vessel, and that the vacuum gauge on the pump and on the retort will register the same.

Experiment II. If the retort be tilted so that the fluid covers the outlet and if a partial vacuum be created in the outlet, the supernatant air in the retort will be under greater pressure than that in the suction bottle, and this unbalanced pressure will force some of the fluid out of the retort. As this occurs the air in the retort may be seen to expand until the vacuum reading in the retort and pump becomes the same, when the flow will cease. Fig. 1.

In order to withdraw more fluid from the retort the partial vacuum within the latter must be overcome by removing the suction attachment, permitting air under atmospheric pressure to bubble into the retort. Suction may then be reapplied when more fluid will be withdrawn. In order to completely empty the vessel, negative pressure alternating with the admission of air must be employed a number of times, depending upon the degree of vacuum, the diameter of the outlet and the relative amount of fluid and air originally contained within the retort.

Experiment III. A Carrel tube sealed at one end and having two small lateral openings was used. Two retorts, one containing fluid and the other empty, were attached to these lateral openings. Suction was then applied to the open end of the Carrel tube, drawing fluid from the retort into the latter. Upon interrupting the suction and permitting air to rush in, it was found that some of the fluid was forced into the previously dry retort. In this experiment the Carrel tube represented the nasal cavity and the two retorts accessory cavities.

The clinical application of this experiment lies in the possibility of drawing pus out of one sinus and by very suddenly changing from negative to atmospheric pressure forcing the pus into a neighboring and unaffected sinus.

In order to reproduce these experiments under conditions almost identical with clinical experience, the following procedure was adopted:

Experiment IV. Roentgenograms of the head of a cadaver were taken to determine the shape and size of the frontal sinus and antrum. A round trephine opening was then made into the external wall of each of these sinuses. A thick flat piece of glass accurately ground to fit each trephine opening was then inserted as nearly air tight as possible (putty or cement). This glass served as a window through which the behavior of fluids within the sinus could be observed under varying conditions. A small secondary accessory opening was drilled into each sinus. Through this opening the sinus could be filled with fluid, after which the opening could be plugged with a stopper. The posterior nares were closed air tight with rubber balloons. The suction nozzle was then applied to the anterior naris. The fluids used were either water, or preferably starch water, which more closely resembles pus in consistency.

Experiment V. Under these conditions, with the head erect and the antrum almost full, suction failed to lower the level of the fluid. If, however, the head were tilted forward and inclined to the opposite side, suction partially emptied the antrum. Upon discontinuing the suction air could be seen bubbling into the antrum to replace the partial vacuum previously induced by the suction. By repeating this process the antrum could finally be practically emptied.

Experiment VI. Frontal Sinus. The outlet of the frontal sinus was partially plugged with cotton, simulating an inflamed mucosa. When the sinus was nearly full of fluid the latter did not gravitate into the nose. With the head erect, upon applying suction part of the fluid was withdrawn. Interrupting the suction, air could be seen entering the cavity. Upon reapplying suction the sinus could be emptied. It will thus be seen that the behavior of fluids in the sinuses was practically identical with that of fluids in the retort.

It may be inferred that to empty the sphenoid it would be necessary to tilt the head forward. The positions of the ethmoid openings are too variable for any general rule. The experiment of drawing fluid out of one sinus by suction and then admitting air to see if the fluid will be forced into adjacent sinus, has not been reproduced entirely to my own satisfaction and requires further trial.

The practical conclusions to be drawn from these experiments are:

First, in order to drain a sinus by negative pressure the head must be placed in the most favorable position—that is, with outlet of the sinus in the most dependent position.

Second, suction should be interrupted to permit air to force its way into the sinus.

Third, the inrush of air after suction may force part of the secretion withdrawn from a diseased cavity into an adjoining sound cavity. This may be obviated by preventing a too sudden inrush of a large volume of air.

Fourth, these experiments do not take into account the pressure due to exudation of fluid in an inflamed closed cavity.

A Modification of the Usual Therapeutic Technic.—The usual method of inducing negative pressure within the nose consists in applying the nosepiece of the suction bottle within the nostril, while the other nostril is being blocked by finger pressure, the patient meanwhile closing off the pharynx by phonating or swallowing. The objections to this method are that continuous suction cannot be maintained, while the closure of the pharynx is often incomplete, and the patient is apt to complain of the disagreeable sensation in the throat and ears during the procedure. As Wells[4] puts it, "The chief drawbacks to the use of ordinary suction are, first, the force necessary to be effective is generally attended with considerable pain, and second, we cannot exclude the eustachian tube from its action." According to the brief statement of Coffin,[5] "The nasal chamber may be closed either by instructing the patient to say Ka-Ka-Ka, or the ballooning of a small rubber bulb in the choanæ."

Most of the objections cited above may be obviated by balloon closure of the choana, and it is concerning this method that I wish to speak of more in detail.

The technic is as follows: The inferior meatus is first cocainized. The finger of a rubber glove makes an admirable balloon. It is inflated to the size of a walnut (using a Politzer bag) and tied off with a cord, having one long and one short end.

To one end of a soft rubber catheter a piece of cord about two feet long is tied. The catheter is then passed through the nose and withdrawn through the mouth, bringing the cord after it. This is simpler than the usual method of fishing the cord out of the pharynx.

The catheter is now detached and the balloon tied to the end of the cord protruding from the mouth. The balloon is then drawn into the choana by traction on the nasal end of the string. It is held by the patient or by the weight of a hemostat.

In addition to this method, I have tried to introduce a long stemmed balloon through the nose and inflating the balloon after it reaches the pharynx. I have also used the Boetscher inflatable postnasal bag, but neither of these methods is as satisfactory as the one described above.

With a balloon in place, one can induce continuous negative pressure in the nasal chamber with but little discomfort to the patient. Sensible children will also tolerate it. My youngest patient was a boy, aged ten years, who received numerous treatments with much benefit. A girl, 16 years old, suffering from pronounced ethmoiditis, recovered after fourteen treatments, although at first it appeared to be an operative case.

The treatment may be prolonged over a considerable period, and the patient can tolerate a higher vacuum up to twenty or twenty-five inches of mercury. Suction may be alternated with air admission as desired.

In conclusion, it may be stated that this method greatly enhances the value of negative pressure in the treatment of sinus disease.

701 RACE STREET.

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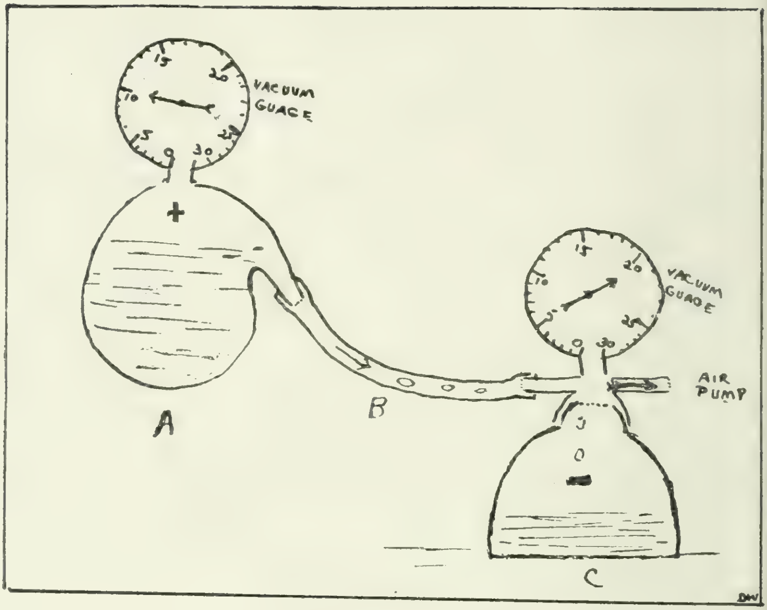

FIG. I

Fig. 1. A represents a paranasal sinus partially filled with fluid. B represents the nasal chamber. C, a suction bottle applied to the nostril. When a partial vacuum is created in C, a vacuum is also created in A, but at first a lesser vacuum than in C, as indicated by plus and minus signs. The fluid will be forced out of A by the unbalanced air pressure until the vacuum readings in A and C become the same, when the flow will cease. In order to get any more fluid out of A it will be necessary to allow air to enter A and then to apply suction as before.

VI.

A NEW LOCAL ANESTHETIC FOR NOSE AND THROAT WORK.

By Albert E. Bulson, Jr., M. D.,
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As chairman of the committee appointed by the Section on Ophthalmology of the A. M. A. to study local anesthesia in ophthalmic work, I was asked by the Council on Pharmacy and Chemistry of the A. M. A. to submit to the members of the committee some samples of butyn, a new local anesthetic manufactured by the Abbott Laboratories, with a view of determining and reporting upon its efficiency and possible advantages in clinical ophthalmic work. The result of the work done by the committee and the conclusions formed are found in a communication published in the Journal of the A. M. A. Feb. 4, 1922. White butyn was being tested clinically in eye work it also was being tested rather thoroughly by the writer and his associates and assistants in nose and throat work, and it is the latter phase of the question which brings forth this communication.

According to the statement of the manufacturers, which practically is confirmed by the Research Committee of the Council on Pharmacy and Chemistry of the A. M. A., butyn is a synthetic anesthetic chemically known as paraaminobenzoyl gamma di-n-butylamino-propanol sulphate. It is produced in powder form, has a bitter taste, and is very soluble in water. Professor Sollman of the Research Committee of the A. M. A., says that butyn is two and one-half times more toxic than cocain when injected hypodermically into albino rats, and Professor Hatcher, of the same committee, says that the fatal dose of butyn when injected intravenously into cats is about equal to that of cocain, though sublethal doses are more dangerous than with cocain. Notwithstanding these findings concerning toxicity, Sollmann further states that the results secured by laboratory tests indicate that butyn may take the place of cocain in whole or in part for surface anesthesia of mucous membranes, and that it may be superior for such purpose to other synthetic anesthetics since it can be used in materially lower concentrations, presumably because of lower absorption. On the other hand, he states that butyn does not appear promising for injection, since the toxicity is materially greater than that of cocain.

In view of this report concerning toxicity, as determined by animal experiments and verified by the animal experiments conducted by our committee, the use of butyn in nose and throat work was undertaken with caution, as it was recognized that the nasal mucous membrane with its greater area and increased absorbing surface, as compared to the conjunctiva, made it advisable to begin with weak solutions and use smaller amounts until the toxicity in the average human could be determined. Therefore, at first butyn in 1 per cent solution was applied once over small areas within the nose, and tests for anesthesia made subsequently at one to three minute intervals. These tests indicated a mild surface anesthesia produced within one minute. Later these tests were extended to include surface anesthesia sufficient for everything pertaining to an examination, including the use of applicators and eustachian catheters, as also for the allaying of the discomfort occasioned by the application of astringents or escharotics. Still later butyn in 2 per cent solution, by repeated application, was employed routinely in all conditions within the nose and throat where surface anesthesia is desired. Finally the strength of the solution was increased to 5 per cent with a view of securing anesthesia for all of the major intranasal operations and testing out more completely the penetrating effect of the anesthesia as also the possibilities of bringing about toxic manifestations.

In the eye butyn produces hyperemia of the ocular and palpebral conjunctiva, more marked in some individuals than others, and more marked in pathologic eyes or those previously the seat of pathologic lesions, though this hyperemia is controlled readily by epinephrin. In the nose and throat a similar hyperemia is produced, but it is less noticeable than in the eye and less objectionable. As butyn produces no ischemic effect there is no shrinking of tissues following its use, such as follows the use of cocain solutions, hence the condition of the intranasal tissues remains approximately the same except for the anesthesia. This is a valuable feature in those cases where a portion or all of a turbinate is to be removed.

I now believe that butyn combined with epinephrin produces a more profound anesthesia than when the butyn is used alone, and the combination has the added advantage of furnishing a relatively bloodless field as well as limiting the absorption of and decreasing the chances of toxic effect of the butyn.

While solutions of butyn in varying strengths of from 1 to 5 per cent have been used, yet it has been found that a 2 per cent solution is sufficiently effective in the ordinary cases, and in view of the possible toxic effects it is considered unnecessary to use the stronger solutions if the milder will do the work. However, for several months a 5 per cent solution combined with epinephrin has been used routinely in major operations within the nose and throat, with very satisfactory anesthesia, and, up to date, without the slightest toxic effects. It is conceivable that in the throat, where absorption is less marked than in the nose, a stronger solution may, after more extended experience with the anesthetic, prove preferable.

Extended use of butyn in nose and throat work finally led to a rather fixed routine in securing satisfactory local anesthesia. For intranasal operation, a 5 per cent solution of butyn combined with epinephrin is swabbed over the operative field and well beyond if possible. Within two or three minutes following the first application, flattened pledgets of cotton, soaked with the solution, but from which the surplus has been squeezed out, are applied snugly over the operative field and whenever possible over the source of sensory nerve supply. These pledgets are removed at the end of five to eight minutes. Five minutes later operative procedures may be commenced with the assurance that in practically all cases the patient will suffer no pain or even discomfort. The exceptions are rare, and comparable to the exceptions under the most thorough efforts to secure anesthesia from cocain, where occasionally we encounter a case of either unusual sensitiveness, or antagonism to the anesthetizing effect of the anesthetic through psychic or other disturbances, and pain and discomfort in such cases occurs irrespective of the agent or technic employed in attempting to anesthetize the tissues. To offset the fear and nervousness that occasionally is present the patient is given a preliminary hypodermic of morphin and atropin, or morphin and hyoscin, but this practice is a routine procedure when cocain is used.

For the ordinary throat operations, including adenectomy, but omitting tonsillectomy which has not been tried under surface anesthesia alone, the practice has been to make about four applications of a 5 per cent solution of butyn to the operative field and well beyond, due care being observed to prevent any of the solution from being swallowed by the patient. For laryngeal operations the applications are made directly into the larynx, though the faucial mucous membrane is swabbed at least once in order to do away with the irritability.

Up to the present time I have had a limited experience with butyn in infiltration anesthesia, but so far it has proven very satisfactory and without toxic effects. I have evidence from several dentists to the effect that infiltration anesthesia with butyn is not only safe but very satisfactory, and it is a well known fact that dentists use infiltration anesthesia extensively and for purposes that really test the efficiency of the anesthetic agent used. In producing infiltration anesthesia I have employed butyn in one-half and 1 per cent solutions, the former being preferable on account of lessened risk of toxic effects.

Under butyn anesthesia, obtained in the manner described, nearly all of the major throat and intranasal operations have been performed, including submucous resections, removal of tumors, turbinotomies, opening of all of the accessory sinuses intranasally, exenteration of the ethmoid cells, removal of adenoid tissue from the nasopharynx, tonsillectomies and removal of benign tumors from the larynx. While all of these operations have been performed under butyn anesthesia during the last few months, many of them, like submucous resections, may times, in no instance, as already stated, has the patient complained of severe pain, and in nearly all instances the patient has not complained of even real discomfort from the operative procedures. Thus in forty-four consecutive submucous resections of the septum, and twenty-three consecutive intranasal operations on the accessory sinuses, 5 per cent anesthesia being employed, the patient in each instance, upon being questioned, declared that the operative procedures had been without pain.

Satisfactory anesthesia for major operations lasts from thirty to forty minutes, oftentimes longer, and in most cases a fair anesthesia will continue for over an hour. The solutions keep well when not exposed to air, and boiling does not seem to impair their anesthetic efficiency.

In no instance, including the hundreds of times that butyn has been used for minor operations and in eye work, have the slightest toxic manifestations been noted. Considering that this means that in the major operations, where a larger quantity of the drug is required and the absorbing surface is larger, no toxic symptoms have been noted, and that in the same cases had cocain been employed in the usual manner, several instances showing more or less toxic symptoms would have occurred, butyn seems to present evidences of superiority. Furthermore, the anesthesia produced by butyn is in the main deeper and more profound than with cocain, and if anything more lasting. This has been noted particularly in accessory sinus work and in the removal of adenoid tissue. Anyone who has attempted to remove adenoid tissue from the young adult under cocain anesthesia, even when a 10 per cent solution is used, knows how infrequently anything like a satisfactory anesthesia has been secured, as he also knows that the use of cocain for that purpose is accompanied by considerable risk of toxic effects.

Concerning the toxicity it is pertinent to remark that butyn has now been used thousands of times by other clinicians, with several of whom I have compared notes, and by many dentists who usually use anesthetics with less than the usual caution, and up to the present writing not a single case of toxic disturbance has been reported. This includes the cases in which butyn has been used experimentally for infiltration anesthesia. It also includes many cases in which solutions of 5 per cent and even stronger have been used rather than the 2 per cent which in all probability will be found sufficient for all ordinary operative work that will be attempted under local anesthesia.

The question arises as to the value of animal experimentation in determining the toxicity of butyn. If the drug is two and one-half times as toxic as cocain, or, in other words, if a 2 per cent solution is equal in toxicity to a 5 per cent solution of cocain, why is it that the free use of a 2 per cent solution of butyn in thousands of cases has not produced a single report of toxic disturbance? Certainly had 5 per cent cocain been used in the same cases and in the same manner sufficient to produce operative anesthesia, quite a number of at least mild toxic cases would have been developed. As a matter of fact, as already stated, 5 per cent butyn has been used by the writer many times for major intranasal operations without noting the slightest signs of toxic disturbance. However, it is very probable that if butyn ever is used as extensively as cocain, there will be cases of toxic effects reported, and then it is a question to decide whether the symptoms are due partly to psychic causes, to idiosyncrasy or to error in using more of the drug than required to produce the desired effect.

Aside from the remote and perhaps questionably possible toxic effects from butyn anesthesia secured in the manner suggested, certain facts concerning this new anesthesia seem to have been clearly established:

1. It is more powerful than cocain, a smaller quantity being required.
2. It acts more rapidly than cocain.
3. Its action is more prolonged than that of cocain.
4. It is less toxic in the quantity required.
5. It is slightly antiseptic.
6. It is less irritating than cocain.
7. It causes no shrinking of tissues.
8. It produces no drying effect upon tissues.
9. It has no ischemic effect.
10. It can be boiled without decomposition.
11. It is nonhabit forming.
12. Its purchase requires no narcotic blank.

406 West Berry St.

VII.

A STUDY OF THE SCHWABACH TEST IN ONE HUNDRED CASES.*

By Robert Sonnenschein, M. D.,

Chicago.

The study here presented was made on 100 unselected cases in the University Nose and Throat Clinic of Professor Gerber of Koenigsberg in 1909. These were examined in detail by means of all the usual functional tests. The result of the analysis of the Weber test (read before the Chicago Laryngological and Otological Society in February, 1911) and of the Rinne test (accepted as a thesis by the American Laryngological, Rhinological and Otological Society, June, 1921) have already been published, and I will take the liberty of quoting somewhat from these papers. The writer believes that the great importance of the functional testing of the ears warrants the reporting of detailed findings.

Inquiries addressed to prominent European otologists at the time examination of the cases was in progress gave the following information regarding the forks employed and the region of the head on which they were placed. While we are perhaps accustomed to regard the Schwabach test as made by holding the fork in the midline of the vertex, many of the otologists quoted use the mastoid processes (just as in the Rinne) or the forehead instead of the vertex. In this country Randall and others employ the Gardner-Brown modification with the fork at the nasal root and the head bent backward.

Panse (Dresden) employed the a^1 fork (435 v. d.) on the mastoid process; Heiman (Warsaw) uses the weighted C (64 v. d.) and c (128 v. d.) fork, usually on the vertex; Schmiegelow (Copenhagen) uses a^1 (435 v. d.) on the vertex; Urbantschitsch (Vienna) uses C (64 v. d.), c^1 (256 v. d.) and c^2 (512 v. d.) on the mastoids; Hartmann (Berlin) applies c (128 v. d.) on the mastoids; Alexander (Vienna) uses the weighed c¹ (154 v. d.) either on the forehead or the mastoid; Politzer (Vienna) always uses c¹ (256 v. d.) on the mastoid; Moeller (Copenhagen) stated that he used a¹ (435 v. d.) applied to each mastoid; Denker (Erlangen) and Siebenmann (Basel) applied forks A (108 v. d.) and a¹ (435 v. d.) on the vertex; Ucherman (Christiania) used the unweighted c¹ (256 v. d.) on the mastoids; Bruehl (Berlin) uses c (128 v. d.) on the vertex; Passow (Berlin) rests c¹ (256 v. d.) on the vertex; Kuemmel (Heidelberg) uses c (128 v. d.); Lucae (Berlin) employs the weighted c (128 v. d.), both placing the fork on the mastoid. Of the fifteen otologists mentioned, we see that eight usually place the fork on the mastoid, six rest the fork on the vertex and one (Alexander) chooses either vertex or mastoid. This at first seems surprising, for we were under the impression that most otologists were making the Schwabach by placing the fork in the midline of the vertex just as in the Weber test. It then occurred to us that it might be wise to note the relation between the results obtained via vertex, forehead and the mastoid.

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*Read at meeting of American Academy of Ophthalmology and Otolaryngology in Philadelphia, October 19, 1921.

The three forks used by us in making the Schwabach test and also the Weber and Rinne were Edelmann's A (108 v. d.); second, the weighted c¹ (154 v. d.). [N. B.—Without weights this fork has the tone of c¹ (256 v. d.), but when weighted as used, has the tone of d sharp (154 v. d.); and, lastly, the small a¹ fork (435 v. d.), (v. d. meaning double vibrations as distinguished from single vibrations. The A and the weighted c¹ forks were excited by allowing them to drop of their own weight and height from a perpendicular to a horizontal position and striking the knee. The a¹ fork was held at right angles to the body with the flat surface of the prong uppermost, and a small pleximeter, such as is used by neurologists for testing the patellar reflex, was allowed to drop of its own weight and height from a perpendicular position directly down upon the flat surface of the prong, thus giving a uniform excitation of the fork in all cases.

Schwabach found in conduction impairment due to diseases of the external and middle ear that a tuning fork applied to the bones of the head was heard longer than normal, and that in cases of auditory nerve affection the perception of the fork was diminished. For this observation Schwabach advances no physical basis, but Steinbrugge believes that the difference in the perception time for the tuning fork sounds in air and bone conduction is probably due both to impairment within the region of sound conduction (including the labyrinthine part) and to the increased, normal, or diminished irritability of the nerve. In our own tests, in order to make them more objective, the average duration of vibration of the forks employed was determined by examining quite a number of normal cases. Then the difference between these figures and the ones obtained by testing other cases showed whether lengthened or shortened bone conduction was present.

In many textbooks the rule is laid down that this test is to be made by holding the fork on the examiner's head (when prolonged bone conduction is suspected), and when no longer heard, placing it on the patient's head and noting the difference in time. If shortened bone conduction is suspected the fork is first placed on the patient's head, and when no longer heard applied to the examiner's head and the difference in time again noted. Of course this method can only be used when the examiner has determined that his own bone conduction is normal. We, however, as aforesaid, have used the more objective method.

Even in normal individuals bone conduction varies, depending upon age (at or after middle age bone conduction is considerably diminished), although it may differ in persons of the same age; the thickness of the cranial bones, the size of the air spaces, such as the mastoid cells, as well as certain anomalies of the skull, depressions, traumatic changes, etc. According to Wanner and Gudden, adhesions between the dura and the bone as well as other traumatic or pathologic changes influence the Schwabach by giving a greatly shortened bone conduction in spite of normal hearing via air. The amount of the hair, the tension of the skin, contact with the auricles and the pressure with which the force is applied, all these may cause variations of the bone conduction. Politzer believes that the Schwabach alone is only in rare instances of great value in differentiating between middle and internal ear disease. When, however, the bone conduction is found prolonged, and the Rinné is decidedly negative, the Schwabach aids in making the diagnosis of interference of the sound conducting apparatus; and where the Schwabach is shortened, together with a positive Rinne and impaired hearing, it greatly assists in the diagnosis of an auditory nerve affection. Politzer also states that "as a prognostic sign it is of considerable importance, because cases with lengthened bone conduction are usually more favorable as to their course and the result of treatment than where it is shortened." This dictum, however, we must modify by stating that in typical otosclerosis, where the bone conduction is greatly prolonged, the prognosis in every direction is poor.

Boenninghaus employs a c^2 fork and cautions against using forks much lower than this, as their heavy vibrations cause confusion in the minds of the patient in differentiating between tactile and auditory perceptions. He does the Schwabach by holding the fork against the mastoid in the region of the fossa mastoidea, with a firm contact (short of producing pain), and then periodically increasing and lessening the pressure, "thus producing an intermittent stimulation which does not fatigue the ear and at the same time facilitates the determination of the maximum stimulation (Reizschwelle)." If the sound is referred to or is said to be heard louder or entirely in the opposite ear, then of course the bone conduction in the examined ear cannot be determined; but indirectly an idea is obtained regarding this ear when the other is examined. If in the second ear the bone conduction is shortened it is still shorter in the first one; if it is normal in the second ear it is shortened in the first one; but if it is lengthened in the second ear, it really shows nothing definite about the first ear, which may have a shortened, normal, or even a prolonged bone conduction, although of course not so prolonged as in the second ear. In comparing the patient's bone conduction with that of the examiner, a difference of a few seconds with forks of long vibration time is of no significance. This may be due to several factors, such as unequal pressure in applying the forks as well as placing it on the identical spot on each mastoid; to a difference in the attention on the part of two individuals; or to variations in the thickness of the bone. One must avoid touching the auricle with the fork, as this causes the former to vibrate and give hearing by air conduction and thus prevent the determination of the moment when the bone conduction ends. N. B., Boenninghaus: "When bone conduction is neither much prolonged nor decidedly shortened, contact of the fork with the auricle will again cause perception of the bone after it can no longer be heard via mastoid."

Bezold determines bone conduction at two pitches, employing the A and the a¹ fork (or others near them in the scale). After the forks are vigorously struck, they are placed on the vertex. Since the other ear cannot be excluded completely from hearing he considers it impracticable to test via mastoid process. "The Weber test will show in which ear the sound is heard the louder." With conduction apparatus interference, the sound is much more prolonged with A (108 v. d.) than with a¹ (435 v. d.); in fact, it often happens that the Schwabach test carried out with a A fork shows a lengthening of bone conduction, whereas the a¹ fork shows a shortening; in such cases disease of the internal ear is also present. Bezold also calls attention to the fact that slight differences in hearing the fork are of no significance. With the low forks usually employed in the Schwabach test, we must bear in mind the powers of observation and concentration on the part of the patient, and also the many adventitious sounds in our environment resembling those of the forks.

It is Mach's theory that the bone conduction is increased by any disturbance in the external or middle ear because the normal outward flow of sound is thereby hindered and a second reflection of sound towards the labyrinth produced. This presupposes, first, a direct or regular transmission from bone to inner ear, and secondly, that normally the conduction apparatus transmits sound vibrations as easily outward as inward. The first assumption is not proven; and, according to Helmholtz, the second theory is untenable because the lever action of the conducting chain, whose long arm consists of the radiating fibers of the drum membrane, easily transmits inward the slightest changes in air vibration, but cannot by means of the short lever arm, namely, the fibers of the ligamentum annulare, transmit them outward. Weber, Brunner, and Lucae believe that the increase of bone conduction in middle or external ear obstruction is partly due to the resonance of the imprisoned column of air. The presence of fluid on either side of the drum membrane increases the bone conduction because the fluid is a better medium for transmission of sound from the bone to the membrana tympani than is the air. The theory of resonance produced by the air in the middle ear spaces does not, however, explain the occurrence (so very often noted) of increased bone conduction (negative Rinne), when the middle ear is filled with secretion. Bezold's theory is based on certain physiologic findings and experiments. For the proper sound transmission via air the conduction apparatus must be in a state of equilibrium, a slight disturbance of which, such as a pure tubal occlusion, is sufficient to produce a decided diminution in air conduction, and an equally definite increase in bone conduction. This equilibrium is likewise affected by large perforations through the drum membrane with a loss of some of the radiating fibers, and a resulting overaction of the tensor tympani muscles. Sclerotic processes in the tympanic property may also cause an increased tension in the conduction apparatus. Bezold holds that bone conduction is brought about by means of the sound conducting apparatus of the middle ear and differs from air conduction only in the fact that with bone conduction the sound waves strike the edge of the drum membrane and the ligamentum annulare and not the flat surface thereof. By experiment he showed that increased tension in the conducting apparatus and at the same time that of the ligamentum annulare, produced lengthened bone conduction and a diminution of air conduction. On this basis he claims to have a simple explanation for the findings of the Weber and Rinne reaction in the majority of cases of the middle ear affection. The increased tension ("anspannung") at any point in the conducting apparatus reduces the ability to transmit air borne sound waves, but heightens its power to conduct vibrations via bone to the labyrinthine fluid. Retjo lays emphasis on the rôle which the round window is supposed to play in bone conduction.

O. Beck and others have called attention to the diminution in bone conduction often noted in cases of lues with otherwise good hearing and no aural symptoms. He says that this lowered bone conduction may be found in 80 per cent of syphilitic subjects, and that it appears mostly in the secondary and only infrequently in the first stage of lues. Goeckermann, Barlow and Stokes found the lowered bone conduction test (diminution in conduction of sound by bone as compared with otherwise normal hearing) positive in 78 per cent of known syphilitics in their series. According to these writers the tests agree with the positive or negative diagnosis of syphilis in 67 per cent and disagrees in 33 per cent. The test was also positive in 48 per cent in patients in whom syphilis was apparently excluded. They conclude that the diminished bone conduction has only "a restricted value as a diagnostic aid in lues owing to its high factor of error."

In the tables herewith appended (showing in seconds the length of time the three forks were heard on the vertex, forehead and mastoid), the same classification of cases is retained as in the paper entitled "A Study of the Rinné Test in One Hundred Cases." First, those with positive Rinne in both ears; second, those with positive Rinne in one ear and negative in the other; and lastly, those with negative Rinne in both ears. The cases here listed showed normal ears in 21; chronic otitis media in 25; acute otitis media in 14; auditory nerve degeneration in 15; otosclerosis in 1; chronic adhesive process in 2; otitis externa in 1; chronic tubal catarrh in 12; traumatic perforation in 1; retracted, cloudy or atrophic drum membranes in 12 cases. In the pathologic cases in some instances both, in others only one ear was involved.

BOTH EARS POSITIVE RINNE.

Age, History, Diagnosis, Etc.	Forks Used	Schwabach —Via—		Schwabach Via Mastoids	
		Vertex	Foreh'd	R. Ear	L. Ear
Case 9. Age 55, M. Affectio nervi acustici. Tinnitus and poor hearing.	A	20	16	20	10
	c_1	14	11	18	16
	a_1	0	0	15	14
Case 10. Age 42. F. Chr. adhesive process. No suppuration. Some affectio nervi acustici. Poor hearing.	A	15	13	30	25
	c_1	30	25	40	40
	a_1	10	7	10	9
Case 12. Age 17. F. Rhinitis atrophica. Drums normal. No ear symptoms.	A	60	45	80	100
	c_1	55	40	70	90
	a_1	15	5	35	45
Case 13. Age 21. F. Ot. Med. Acuta (dex) six months ago. Now healed. Tinnitus right.	A	60	40	60	80
	c_1	45	35	50	70
	a_1	15	0	30	35

Age, History, Diagnosis, Etc.	Forks Used	Schwabach —Via—		Schwabach Via Mastoids	
		Vertex	Foreh'd	R. Ear	L. Ear
Case 14. Age 19. F. Epistaxis. No ear symptoms. (Normal.)	A	52	52	60	80
	c_1	48	48	60	60
	a_1	10	10	45	45
Case 17. Age 16. F. No ear symptoms. Normal.	A	35	25	70	90
	c_1	65	55	90	100
	a_1	34	24	33	35
Case 18. Age 20. F. Rhinitis atrophica. Laryngitis chr. Some tinnitus and impaired hearing.	A	35	18	35	75
	c_1	35	25	25	60
	a_1	15	0	30	45
Case 20. Age 15. M. (Normal Ears.) No ear symptoms. Deviatio septi.	A	60	35	90	90
	c_1	40	25	60	60
	a_1	18	0	32	35
Case 23. Age 19. M. Laryngitis tubec. Slight feeling of fullness in ears. Normal ears.	A	25	25	65	70
	c_1	23	23	55	65
	a_1	15	15	35	40
Case 25. Age 22. M. Ears normal. No symptoms.	A	35	40	70	70
	c_1	30	35	55	55
	a_1	15	18	38	35
Case 26. Age 24. F. Nasal polyps. No ear symptoms.	A	30	20	50	50
	c_1	45	30	65	70
	a_1	15	8	40	35
Case 27. Age 50. M. Dev. septi. Some impairment of hearing. (Rather thick hair.)	A	30	30	45	60
	c_1	38	36	50	55
	a_1	10	10	15	15
Case 28. Age 15. M. Atrophic drum membranes. Tinnitus right for two weeks. Some affectio nervi.	A	20	20	40	40
	c_1	25	25	55	55
	a_1	20	20	35	35
Case 29. Age 29. M. Some tinnitus left and impaired hearing after suppuration.	A	30	30	70	60
	c_1	40	40	60	55
	a_1	35	25	45	35
Case 30. Age 56. F. Affectio nervi acustici. Marked tubal catarrh left.	A	50	45	70	65
	c_1	50	35	80	70
	a_1	20	30	45	30
Case 31. Age 79. M. Tinnitus and impaired hearing. Affectio nervi.	A	35	0	0(?)	0(?)
	c_1	40	40	40	40
	a_1	0	0	25	25

(Cannot explain reaction of A fork.)

Age, History, Diagnosis, Etc.	Forks Used	Schwabach —Via—		Schwabach Via Mastoids	
		Vertex	Foreh'd	R. Ear	L. Ear
Case 33. Age 23. M.	A	45	50	70	70
Ethmoiditis. No ear symptoms. Rather thick hair. Hears better via forehead.	c_1	40	40	70	80
	a_1	25	35	55	55
Case 34. Age 32. M.	A	22	20	30	30
Tubal catarrh five years ago. No ear symptoms now. Slight affectio nervi.	c_1	20	18	30	45
	a_1	10	0	20	32
Case 35. Age 23. M.	A	20	30	70	90
Chronic laryngitis. Normal ears. Thick hair.	c_1	25	32	60	72
	a_1	25	18	45	45
Case 36. Age 64.	A	50	45	80	80
Tinnitus.	c_1	40	40	60	70
Tubal catarrh.	a_1	20	25	40	40
Case 38. Age 21. M.	A	10	20	40	65
Tinnitus.	c_1	10	18	45	55
Some affectio nervi.	a_1	10	10	25	40
	(Cannot explain Rinne and Schwabach in this case.)				
Case 41. Age 22. F.	A	45	50	90	80
Chr. ethmoiditis. No ear symptoms. Slight retraction.	c_1	45	55	70	70
	a_1	20	20	35	35
Case 42. Age 30. F.	A	55	60	110	120
No ear symptoms.	c_1	35	35	60	70
Normal drums.	a_1	22	22	45	48
Case 43. Age 21. F.					
Recently tinnitus and impaired hearing. Some atrophy of drum membranes.	A	30	30	70	70
	c_1	32	32	60	60
	a_1	10	10	35	35
Case 53. Age 15. F.	A	40	45	65	65
Adenoids.	c_1	55	55	60	60
No ear symptoms now.	a_1	30	30	50	50
	(Weber to right side, though no apparent lesion.)				
Case 54. Age 27. M.	A	65	60	80	80
No ear symptoms.	c_1	55	55	75	75
Drums slightly cloudy.	a_1	30	30	60	60
Case 56. Age 15. M.	A	50	50	80	90
No ear symptoms.	c_1	45	40	80	80
Nasal polyps.	a_1	35	35	55	55
	(Cannot explain Weber to left side.)				

Age, History, Diagnosis, Etc.	Forks Used	Schwabach —Via—		Schwabach Via Mastoids	
		Vertex	Foreh'd	R. Ear	L. Ear
Case 57. Age 14. F. Chronic rhinitis. No ear symptoms. Thick hair. Hears better on forehead.	A	40	45	90	95
	c₁	40	45	60	70
	a₁	20	25	45	55
Case 59. Age 30. M. No ear symptoms. Some retraction of drums.	A	45	45	85	85
	c₁	40	40	80	80
	a₁	22	22	45	45
Case 60. Age 20. M. No ear symptoms. Normal drums.	A	40	30	75	75
	c₁	45	30	60	70
	a₁	20	20	45	45
Case 62. Age 26. F. No ear symptoms. Drum membranes normal. Thick hair.	A	65	65	95	100
	c₁	48	55	70	70
	a₁	20	35	35	40
Case 64. Age 28. M. Affectio nervi acustici bilat. Tinnitus left ear. Rather thick hair.	A	30	30	55	55
	c₁	25	30	50	50
	a₁	15	20	30	30
Case 69. Age 23. M. No ear symptoms.	A	55	58	80	90
	c₁	50	50	70	80
	a₁	30	30	45	55
Case 70. Age 27. M. Acute laryngitis. Tinnitus, left for two weeks. Some affectio nervi acustici. Rather thick hair.	A	45	50	80	80
	c₁	40	30	50	50
	a₁	20	15	40	35
Case 72. Age 30. M. No ear symptoms.	A	55	75	90	90
	c₁	45	62	80	75
	a₁	50	50	60	60
Case 73. Age 40. F. Nasal polyps. Cloudy drum membranes. Occasional tinnitus. Thick hair.	A	35	50	90	90
	c₁	30	35	60	60
	a₁	20	22	40	40
Case 76. Age 16. M. Chr. Rhinitis and laryngitis. Cloudy right drum and feeling of fullness.	A	50	60	90	90
	c₁	40	40	60	60
	a₁	30	35	50	50
Case 80. Age 12. M. Rhinoscleroma. Some retraction of drums. Slight affectio nervi acustici.	A	45	55	90	80
	c₁	35	38	65	65
	a₁	25	25	40	40

Age, History Diagnosis, Etc.	Forks Used	Schwabach —Via—		Schwabach Via Mastoids	
		Vertex	Foreh'd	R. Ear	L. Ear
Case 83. Age 20. F. Rhin. hyper. Ears normal. Rather thick hair.	A	55	62	90	92
	c_1	45	55	80	85
	a_1	20	30	50	50
Case 85. Age 46. F. Tumor laryngis. Affectio nervi acustici. Thin hair.	A	60	50	80	80
	c_1	40	40	50	50
	a_1	18	18	35	35
Case 91. Age 40. M. Rhinitis atrophica. Some cloudiness drum membranes. No ear symptoms. Moderately thick hair. Some affectio nervi, left ear.	A	45	50	80	80
	c_1	30	35	50	50
	a_1	18	25	35	35
Case 95. Age 32. F. Drum membranes normal. Increasing impairment of hearing. Thick hair. Affectio nervi(?).	A	40	45	50	55
	c_1	25	28	40	45
	a_1	10	10	25	30
Case 97. Age 22. F. Rhinitis hyper. Ears normal. Thick hair.	A	50	63	95	95
	c_1	35	45	65	65
	a_1	23	18	40	40
Case 99. Age 51. M. Drum membranes normal. Retroauricular abscess left opened 4 years ago. Pain in left ear past few weeks. Bald.	A	60	45	95	100
	c_1	50	40	70	75
	a_1	15	5	35	35
Case 100. Age 73. M. Both drum membranes retracted. "Thumping" in ears and poor hearing past few weeks. Thin hair. Slight affectio nervi.	A	30	40	55	60
	c_1	25	35	50	55
	a_1	0	10	25	25

POSITIVE RINNE ON ONE AND NEGATIVE ON OTHER EAR.

Age. History, Diagnosis, Etc.	Forks Used	Schwabach —Via—		Schwabach Via Mastoids	
		Vertex	Foreh'd	R. Ear	L. Ear
Case 2. Age 17. M. Ot. med. supp. chr. sin. Total destruction of drum.	A	68	55	90 Pos.	100 Neg.
	c_1	58	50	70	75
	a_1	42	35	58	60
Case 3. Age 13. F. Ot. med. supp. chr. sin. Total destruction. Some vertigo.	A	70	65	110 Pos.	70 Neg.
	c_1	55	50	70	60
	a_1	30	28	25(?)	30

Age, History, Diagnosis, Etc.	Forks Used	Schwabach —Via—		Schwabach Via Mastoids	
		Vertex	Foreh'd	R. Ear	L. Ear
Case 5. Age 11. M. Cat. Tubanus dextra.	A	60	60	100 Neg.	90 Pos.
	c_1	55	50	70	80
	a_1	35	31	60	50
Case 7. Age 20. F. Radical mastoid. (Dex.) Cloudy drum (Sin). High tinnitus.	A	30	22	40 Neg.	0
	c_1	18	15	25	0
	a_1	10	5	20	0
Case 8. Age 51. M. Ot. med. Sup. Chr. Dex. Ot. med. Sup. Acuta Sin. Tinnitus right. Heard as long on vertex as mastoid.	A	75	60	60 Neg.	70+
	c_1	55	45	55	55
	a_1	20	14	25	20
Case 16. Age 16. F. Ot. med. Sup. Chr. Dex. No tinnitus, etc. Heard as long on vertex as mastoid.	A	60	30	60 Neg.	70 Pos.
	c_1	70	35	60	65
	a_1	20	12	35	40
Case 19. Age 62. M. Ot. med. sup. acuta sin. Pain in ear. Heard as long on vertex as mastoid.	A	70	45	45 Pos.	30 Neg.
	c_1	65	50	45	85
	a_1	15	15	30	30
Case 21. Age 32. M. Chr. tubal catarrh (left). "Pressure" in ear, but no tinnitus. Heard as long on vertex as mastoid.	A	60	60	65 Pos.	50 Neg.
	c_1	60	60	80	60
	a_1	30	30	40	25
Case 24. Age 49. F. Ot. med. sup. ac. sin. Tinnitus bilateral. Heard as long on vertex as mastoid.	A	80	45	70 Pos.	75 Neg.
	c_1	70	40	60	65
	a_1	22	10	25	30

*N. B. Cases No. 2 and 3 show how independently ears react to Rinne test, one side as compared with other.

†N. B. In this case closure of left ear with finger caused fork to be heard in that ear when placed on vertex or mastoid.

Age, History, Diagnosis, Etc.	Forks Used	Schwabach —Via—		Schwabach Via Mastoids	
		Vertex	Foreh'd	R. Ear	L. Ear
Case 32. Age 40. F. Healed Ot. med. chronica bilateralis. Also affectio nervi. Heard as long on vertex as mastoid.	A	20	18	15 Neg.	35 Pos.
	c_1	30	30	30	60
	a_1	8	8	15	30
Case 45. Age 15. F. Adenoids; atrophic drums. Some impairment of hearing.	A	60	70	105+	100 Neg.
	c_1	50	55	80	80
	a_1	25	30	60	60

Case 46. Age 50. M. Right complete destruction of drum; left retracted. Impaired hearing. Affectio nervi dex. Catarrh tub. sin. but heard as long on vertex as mastoid.	A	35	30	45 Neg.	35 Pos.
	c_1	35	30	30	35
	a_1	10	6	10	10
*Case 47. Age 14. F. Very unintelligent. Bilat. Ot. med. chr. Much destruction. Poor hearing.	A	55	60	70 Pos.	75 Neg.
	c_1	30	40	60	55
	a_1	20	25	25	25

*Cannot explain a_1 in this case.

Case 48. Age 16. M. Acute mastoiditis left, 4 years ago. Past 3 weeks again pus. Right ear normal. Rather thick hair.	A	30	55	115 Pos.	120 Neg.
	c_1	70	85	105	90
	a_1	40	45	50	40
†Case 49. Age 28. M. Laryngitis tubc. No ear symptoms. Rather thick hair, but hears better via vertex.	A	50	30	80 Neg.	100 Pos.
	c_1	55	35	65	75
	a_1	25	18	42	50

†Cannot explain negative Rinn. in right ear.

Case 51. Age 13. F. Ot. med. acuta supp. bilat. Almost healed.	A	65	60	80 Pos.	80 Neg.
	c_1	50	40	70	70
	a_1	40	30	50	60
Case 52. Age 49. F. Tinnitus and poor hearing. Retracted drum membranes.	A	60	70	120 Pos.	130 Neg.
	c_1	40	50	90	105
	a_1	20	12	30	50
Case 61. Age 8. F. Cholesteatoma right with impaired hearing. Left normal.	A	60	60	80 Neg.	85 Pos.
	c_1	45	40	60	70
	a_1	30	30	35	35
Case 63. Age 7. M. Ot. med. chr. sup. sin. Right normal. Child not very intelligent.	A	50	45	70 Pos.	80 Neg.
	c_1	60	60	90	90
	a_1	35	30	60	65
Case 65. Age 32. F. Otitis externa dex. with mild otitis media acuta, Pain right ear.	A	60	50	95 Neg.	90 Pos.
	c_1	65	55	100	100
	a_1	20	20	40	40
Case 67. Age 47. F. Ot. media sup. acuta. Dextrain and impaired hearing. Thick hair.	A	60	65	90 Neg.	90 Pos.
	c_1	50	60	80	80
	a_1	20	25	40	45

Age, History, Diagnosis, Etc.	Forks Used	Schwabach —Via—		Schwabach Via Mastoids	
		Vertex	Foreh'd	R. Ear	L. Ear
Case 71. Age 22. M.					
Traumatic perforation	A	40	40	100 Pos.	90 Neg.
left. Impaired hearing.	c_1	40	30	70	65
Some tinnitus. Thin hair.	a_1	25	20	40	40
Case 74. Age 55. F.					
Healed otitis media chr.	A	40	40	80 Pos.	80 Neg.
sin. Occasional tinnitus.	c_1	35	30	70	75
Subacute otitis med. dex.	a_1	22	12	85	60
Case 75. Age 45. M.	A	80	80	90 Pos.	100 Neg.
Otitis media acuta sin.	c_1	70	70	70	80
Pain left ear.	a_1	25	20	35	45
Case 77. Age 19. F.					
Radical mastoid right 2	A	50	35	70 Neg.	90 Pos.
years ago. Hearing very	c_1	45	35	60	70
poor. Left ear normal.	a_1	25	20	35	45
Case 78. Age 16. F.	A	70	60	90 Neg.	80 Pos.
Ot. med. subacuta supp.	c_1	50	40	80	65
dex. Left ear normal.	a_1	35	25	50	50
Case 81. Age 49. M.					
Healed Ot. med. acuta	A	30	40	90 Neg.	90+
dex. Ot. med. non-supp-	c_1	40	40	80	80
acuta sin. Still some hear	a_1	25	20	50	50
ing impairment right.					
Case 84. Age 13. M.					
Otitis med. acuta sin.	A	50	60	90 Pos.	90 Neg.
Right ear normal.	c_1	40	45	90	80
Rather thick hair.	a_1	35	40	60	60
Case 86. Age 11. M.	A	80	90	120 Neg.	125 Pos.
Acute tubal catarrh	c_1	70	60	110	120
right. Left normal.	a_1	45	55	55	55
Case 87. Age 51. F.					
Otitis med. supp. ac. dex	A	35	50	90 Pos.	75 Neg.
2½ years ago. Otitis med.	c_1	35	45	70	60
supp. subacuta sin. Mod-	a_1	25	38	52	45
erately thick hair.					
Case 88. Age 14. F.					
Large adenoids. Tinnitus	A	20	35	80 Pos.	50 Neg.
left. Left drum retracted.	c_1	18	30	68	68
Thick hair.	a_1	15	25	53	50
Case 89. Age 26. F.					
Dry perforation right,					
following ot. media supp.	A	60	70	105 Neg.	95 Pos.
in childhood. Tinnitus	c_1	50	65	75	70
right. Left drum cloudy	a_1	20	30	45	35
and retracted. Thick hair.					

Age, Diagnosis, History, Etc.	Forks Used	Schwabach —Via— Vertex	Foreh'd	Schwabach Via Mastoids R. Ear	L. Ear
Case 90. Age 46. F.					
Right drum retracted.	A	60	70	120 Pos.	110 Neg.
Total destruction left drum.	c_1	45	60	75	70
Tinnitus and poor hearing left. Moderately thick hair.	a_1	25	18	45	38
Case 92. Age 54. F. Unhealed radical mastoid left. Pains left side.	A	25	35	65 Pos.	55 Neg.
Right normal except cloudiness.	c_1	10	20	40	30
Apparently totally deaf left side. Thin hair.	a_1	0	0	20	10
Case 96. Age 24. F.					
Right normal. Ot. media	A	50	60	90 Pos.	80 Neg.
supp. chr. sin. Rather thick	c_1	35	42	60	50
hair.	a_1	18	25	38	35
BOTH EARS NEGATIVE RINNE.					
Case 1, Age 11. M.	A	60	50	110	120
Ot. med. chr. supp. after	c_1	55	45	85	100
scarlatina.	a_1	30	25	60	70
Case 4. Age 16. F.					
Ot. med. chr. s. bilat.	A	80	68	90	80
Fetid pus. Attic fistula	c_1	75	65	80	70
left.	a_1	35	28	58	46
Case 6. Age 34. F.					
Right drum normal. L.	A	50	40	55	80
shows catarrh. A typical	c_1	25	18	35	55
otosclerosis with nerve involvement. Tinnitus.	a_1	8	4	15	25
Case 11. Age 32. F.					
Affectio nervi acustici.	A	45	40	44	50
Right drum cloudy. Left	c_1	50	45	55	65
drum normal. Poor hearing and tinnitus.	a_1	7	4	12	15
Case 15. Age 11. M.	A	50	40	65	75
Subacute secretory ca-	c_1	45	30	70	80
tarrh. Bilateral. Tinnitus.	a_1	28	25	60	65
Case 22. Age 45. M.					
Retracted drum mem-	A	100	85	100	85
branes. Tinnitus and some	c_1	60	60	75	60
impairment of hearing.	a_1	35	35	35	35
Case 37. Age 15. M.					
Right—Intermittent ot.	A	65	30	120	100
media chr. Left—unhealed	c_1	90	80	100	100
radical mastoid. Rather thick hair.	a_1	50	30	55	50

Age, History, Diagnosis, Etc.	Forks Used	Schwabach —Via—		Schwabach Via Mastoids	
		Vertex	Foreh'd	R. Ear	L. Ear
*Case 39. Age 17. M.					
Ot. media chr. in child-	A	100	100	120	120
hood. Right drum retract-	c_1	90	90	90	90
ed. Tinnitus. Rhin. hyper	a_1	40	40	55	55
& adenoids. Affectio nervi.					
Case 40. Age 17. F.					
Ot. med. s. acuta bilat-	A	45	45	65	75
eralis. Earache. Impaired	c_1	60	60	75	75
hearing.	a_1	20	20	35	45
Case 44. Age 35. F.					
Ot. med. chr. bilat. in	A	45	45	100	90
childhood. Now dry. Large	c_1	65	65	100	90
perforations.	a_1	30	30	50	45
Impaired hearing.					
Case 50. Age 9. F.	A	40	40	40	40
Ot. media chronica bilat.	c_1	60	60	60	60
Much destruction.	a_1	45	45	50	50
Case 55. Age 18. F.					
Ot. med. supp. chr. bilat.	A	10	20	50	60
Tinnitus and impaired	c_1	25	30	55	65
hearing.	a_1	30	30	50	55
Case 58. Age 18. M.					
Ot. med. supp. chr. bilat.	A	35	25	70	70
Attic fistula left. Poor	c_1	20	20	60	60
hearing.	a_1	22	20	35	40
Case 66. Age 15. M.					
Ot. med. sup. chr. bilat.	A	65	60	110	100
Impaired hearing. Mas-	c_1	75	70	95	90
toid operation left side.	a_1	40	30	60	60
Case 68. Age 14. M.					
Ot. media supp. acuta	A	70	80	90	90
dextra. Ot. med. supp.	c_1	40	45	80	80
chronica sinistra.	a_1	35	38	75	80
Thick hair.					
Case 79. Age 16. F.					
Adenoids; rhin. hyper.	A	28	38	65	55
Cloudy drum membranes.	c_1	25	32	62	50
Hearing much improved.	a_1	20	20	65	50
Affectio nervi.					
Case 82. Age 28. F.					
Otitis med. supp. chr.	A	50	80	90	100
Bilateralis. Much destruc-	c_1	45	50	75	75
tion both sides.	a_1	30	40	60	60
Thick hair.					

*Forks on left mastoid and forehead apparently heard in right ear.

Age, History, Diagnosis, Etc.	Forks Used	Schwabach —Via—		Schwabach Via Mastoids	
		Vertex	Foreh'd	R. Ear	L. Ear
Case 93. Age 20. F.					
Large polyps right; total	A	55	65	90	105
destruction left. Suppura-	c_1	55	65	80	90
tion and impaired hearing.	a_1	20	35	40	40
Case 94. Age 32. F.					
Drum membranes cloudy					
and right retracted. Im-	A	65	78	95	115
paired hearing with tinni-	c_1	55	65	80	95
tus in left ear. (Very thick	a_1	20	22	35	50
hair.)					
Case 98. Age 21. F.					
Radical mastoid opera-	A	30	45	55	75
tions both sides. Hearing	c_1	30	40	32	50
very poor.	a_1	15	20	25	42

An analysis of the figures obtained from these tables is as follows: On the vertex the average hearing for A (108 v. d.) was 48 seconds; for the c^1 weighted fork (154 v. d.), 34 seconds; and for the a^1 fork (435 v. d.) was 22 seconds. On the forehead the A fork was heard an average of 47 seconds, the weighted c^1 fork, 43 seconds (!), and the a^1 fork an average of 21 seconds. It is to be noted that the c^1 weighted fork was heard longer on the forehead than on the vertex, whereas the other forks were heard on the average longer on the vertex, despite the presence of hair in many of the cases. The grand average of all the forks showed for the vertex 37.1 seconds and for the forehead 37.3 seconds, or, practically speaking, no difference at all. Taking the averages of the two mastoids together, we find for the A fork an average duration of 76 seconds, for the weighted c^1 fork an average duration of 66 seconds, and for the a^1 fork a duration of 41 seconds, or a grand average of all forks via mastoid of 60.9 seconds.

The ratio between the total average of hearing of the three forks via mastoid and via forehead and vertex is as 60.9 to 37.3 seconds, or actually as 3 to 2. Now we do not postulate that this is always so, but can only state the findings in the series of cases reported. These were examined in a quiet room, much time and care taken to repeat the test several times on the same patient if necessary, often having the patient return a few days later in order to check up the first examination. So that we can state that these tabulations show the actual and accurate findings in these 100 cases. The duration of time that the forks were heard is longer than we usually assume or probably experience, but the only explanation for this is the one we have just given. The weighted c¹ fork was heard longer than the A fork in certain cases, viz.. both ears in cases 15 and 30; one ear, cases 19, 38 and 33; on the vertex and forehead cases, 37, 44, 40 and 66; one or both ears and vertex and forehead, cases 11, 50, 28, 17, 26, 10, 63, 65, 55, 34 and 32; on the vertex only case 81. Normal ears, cases 33, 17 and 26; cases of otitis media chronica, cases 32, 11, 37, 40, 44, 50, 66, 63; cases of otitis media acuta, 19, 65 and 81; cases of tubal catarrh, 30, 15 and 88; cases of affectio nervi austici, cases 30, 31, 11, 10 and 28; chronic adhesive process, case 10. The a¹ fork was heard longer than the weighted c¹ fork in case 74 subacute otitis media; one ear, case 18, some affectio nervi acustici; one ear, case 79, affectio nervi acustici.

The actual hearing of all forks via forehead and vertex was practically the same, often despite the presence of the hair. In 19 cases where the hair was quite thick (cases 33, 35, 57, 73, 83, 91, 97, 67, 84, 87, 88, 89, 90, 96, 68, 82, 93, 94, 98), the hearing via forehead was longer than that via vertex. In 16 cases where there is no special mention of heavy hair or where the hair is thin, the hearing via forehead was longer (cases 38, 41, 42, 25, 72, 76, 80, 100, 45, 47, 48, 52, 86, 92, 55 and 79). Thus in 35 per cent of the cases (19 with thick hair and 16 with thin hair) the forks were heard longer by way of the forehead, but in the other 65 per cent the forks were perceived longer by way of the vertex.

The fork placed on the vertex, or root of the nose (in the Gardner-Brown test), rests largely by its own weight, and the intensity of sound is not so much influenced by pressure used in retaining it in position; but when applied to the forehead, unless the patient's head is bent back to a rather uncomfortable degree, it is difficult to keep the fork in contact with the head without applying considerable pressure.

CONCLUSIONS.

1. On the vertex the average hearing for A (108 v. d.) was 48 seconds; for the c¹ weighted fork (154 v. d.), 34 seconds,

and for the a^1 fork (435 v. d.), was 22 seconds. On the forehead the A fork was heard an average of 47 seconds, the weighted c^1 fork 43 seconds, and the a^1 fork an average of 21 seconds. It is to be noted that the c^1 weighted fork was heard longer on the forehead than on the vertex, whereas the other forks were heard on the average longer on the vertex despite the presence of hair in many of the cases. The grand average of all the forks showed for the vertex 37.1 seconds and for the forehead 37.3 seconds or, practically speaking, no difference at all. Taking the averages of the two mastoids together, we find for the A fork an average duration of 76 seconds, for the weighted c^1 fork an average duration of 66 seconds and for the a^1 fork a duration of 41 seconds, or a grand average of all forks via mastoid of 60.9 seconds.

2. The duration of bone conduction in this series of tests is apparently longer than we have in routine examinations, but this finding may be due to the peculiarly favorable circumstances under which our cases were tested. Slight variations in bone conduction, as noted in doing the Schwabach test, are of no significance, since many factors, such as age, conformation of the skull bones, concentration on the part of the individual, etc., may influence the findings.

3. In this series of cases the ratio between the hearing via mastoids and that obtained by way of the vertex of the forehead was 3 to 2. Despite hair there was in most of the cases (65 per cent) practically no difference in duration of hearing via vertex or forehead.

4. Many noted authorities test bone conduction by way of the mastoid, and it would seem, in view of the results obtained in this series, that the bone conduction factor of the Rinne test could serve as a Schwabach, this eliminating testing by way of the vertex or the forehead except where the Weber test is deemed advisable, and the Schwabach is then done at the same time by the same maneuver.

The A fork (108 v. d.) is a large instrument and cannot easily be applied to the mastoid process, but the weighted c^1 (154 v. d.) is comfortably held either on the vertex, forehead, root of nose or mastoid and could thus be used for the Weber, Schwabach and Rinne tests. There is really a difference of only 46 vibrations between the A fork (108 v. d.) and the weighed c¹ (154 v. d.) which, when weighted, gives a tone of d sharp, but there is a great difference in the weight and size of the fork.

5. The suggestion regarding the possible elimination of the A fork and use of the wighted c¹ fork may be of some value to those men who do not wish to, or are unable to obtain many forks for routine work. The writer himself is very fond of the A fork for use in the Weber and Schwabach tests where the latter is done by way of the vertex, but he simply mentions this fact for the reason above stated. The a¹ fork (435 v. d.) is most valuable, not only in doing the Rinne, but also in testing hearing of the voice by air conduction. Since its tone lies in the socalled "speech area" (designated by Bezold as extending from c¹ to g²), it is assumed that if not thus heard there is no hearing for the ordinary range of speech. (N. B.—It is known that some of the harmonics of the sounds employed in speech may be much higher than those mentioned—e. g., the fifth harmonic of the vowel e has about 3,000 vibrations).

6. The question of the part played by the round window in bone conduction is a very important one and will require careful study.

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VIII.

TONSILLECTOMIES IN ADULTS FOR RHEUMATISM WITH CRITICAL REVIEW OF RESULTS.*

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Much that has been written of the tonsil operation deals with the surgical technic. Comparatively little has appeared in laryngologic literature dealing with clinical problems or the results from the patient's standpoint.

The clinical problems are, of course, not strictly within the province of the laryngologist, but the operator cannot and should not, it seems to me, lay aside his share of responsibility for the results. The overzealousness of a large part of the medical profession and the general public, too, in urging tonsillectomies, is tending at this time to bring disrepute on the operation. The duty of safeguarding it lies to a large extent with the laryngologist. The past history of operations in other surgical fields certainly impresses one with the need of greater care in selection of cases for tonsillectomy, lest the operation be greatly abused. These considerations prompted the writer to review his own (adult) tonsillectomies. A review of this kind is admittedly imperfect and far from satisfactory, but it may prove of value as a small contribution to the larger and better mass of evidence that is gradually accumulating on the tonsil question.

LIMITATIONS.

First—All adult cases in which tonsillectomies were done solely for ear, nose and throat diseased conditions are excluded, such as cases of quinsy, accessory sinus disease, middle ear disease, recurrent laryngitis and bronchitis, etc. Cardiovascular cases are excluded.

Second—Cases that date back over six years are excluded, for two reasons. First, it has been only the past six years that the writer has been impressed with the necessity of using greater care in the selection of these operative cases, by doing group study, with the combination of careful examinations by competent internists, orthopedists and dentists. Second, cases that date back over a long period of time are excluded because so many other factors may enter into the question of improvement or deterioration in the health of these adult invalids.

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Third—All cases are excluded that are of shorter duration of observation than four months, for it is a common experience to see marked temporary improvement follow a tonsillectomy, say for a few weeks, with recurrence of the symptoms. This seems especially true of rheumatic cases. Such cases are often lost sight of after the tonsil wounds are healed, and the subsequent results are apt to be unknown. It has been suggested that the enforced rest of the intestinal tract after a tonsillectomy has much to do with this temporary improvement. It has also been suggested that the effect of general anesthetic on the muscle spasm, more or less constant in some rheumatic cases, is decidedly beneficial for a short period of time.

These limitations left 130 cases out of 477 adult tonsillectomies. In the 130 cases, patients suffered from and sought operation because of rheumatism; most of them with impairment of general health; some, however, with no impairment of health. It seemed best to the writer to limit this report to this group of rheumatic cases, because the indications for operation were evident on account of the pain and stiffness common to all cases of this group. One could depend more upon the accuracy of the subsequent results as determined by examination of the patients, and checked up by questionnaires which were answered by all except 29 of the patients. No operation was done in any case of definite rheumatoid arthritis. No tonsillectomy was done during an acute attack of rheumatism with fever. The selection was made from patients who were not entirely bedridden, most of them confined to bed during an acute exacerbation. Many suffered pain and stiffness only periodically. Some were more severe, requiring the use of crutches or rest in bed or some immobilizing apparatus; a few had joint operations.

TABLE I.

Age	Cases
19 to 30	20
30 to 40	50
40 to 50	38
50 to 60	18
60 to 65	4
Total	130

Eighty-eight of these cases were between the ages of 30 and 50.

TABLE II.

Duration of rheumatism	Cases
1 to 6 months	26
6 months to 2 years	38
Over 2 years	48
Indefinite time or not stated	18
Total	130

TABLE III.

Parts Affected	Cases
Shoulder	51
Arms	24
Hands	43
Elbow	5
Neck	9
Back	11
Hip	11
Leg	14
Knees	37
Feet	17
Ankle	14

It is noteworthy that the shoulder joint was affected more frequently than any other joint. Owing to the anatomic construction of the shoulder joint, it is probable that it is more subject to injury from strains than other joints. It is believed that injury to a joint is frequently a forerunner of the localization of infection in that joint. The orthopedic and X-ray examinations frequently were negative, so far as making out definite bony or cartilaginous changes, in many patients, even though they were chronic sufferers from pain and stiffness.

TABLE IV.

Character of rheumatism with results of tonsillectomy:

	Cases	Imp.	Much Imp.	Cured	Not Imp.	Unk'wn Results
Chronic Arthritis—						
Mild	69	13	7	22	12	15
Moderate	28	6	4	11	5	2
Severe	4	0	0	0	4	0
Acute arthritis	3	0	1	2	0	0
Neuritis	6	1	0	0	2	3
Myalgia	20	3	2	5	1	9
Total	130	23	14	40	24	29

Of the severe and moderately severe cases, many showed bony or cartilaginous changes. In these cases where secondary foci were chronically established, results were not only more slowly obtained, if obtained at all, but complete cure was rare. It was the opinion from group study of the mild and moderately severe arthritic cases that the joint condition was a chronic infection of the synovial membrane or of the capsule of the joint. The X-ray examination was negative in cases, even though effusion existed.

TABLE V.

Throat History	Cases
Attacks of tonsillitis	54
Quinsy	9
Soreness	23
No tonsil complaint	46

In the forty-six cases where there was no history of tonsillitis or even soreness it is, of course, more than probable that in early life there had been definite attacks of tonsillitis. It is likely that a chronic infection dates back to a definite acute attack, the tonsils being streptococci carriers from that time on. How early in life a chronic infection begins is a problem in itself. The writer some time ago in examining histologically some apparently normal tonsils in very young children, searching for signs of tuberculosis, was surprised to find streaks of chronic inflammation with well defined fibrosis here and there in many sections of the tonsil tissue. Some of these children had no history indicative of a chronic tonsil infection, but were operated on solely because of hypertrophied tonsil and adenoid.

TABLE VI.

	Cases
Abscessed teeth removed before tonsillectomy	26
Colectomy	1
Previous incomplete tonsil operations	12

TABLE VII.

Subsequent history:

	Cases
Rheumatism, improved	25
Rheumatism, much improved	15
Rheumatism, cured	40
Rheumatism, not improved	24
Unknown results	26
Total	130

The subsequent results are based on personal examination of the patients by the writer, by the internists or by the orthopedist, and checked up by questionnaires received from all of the 130 cases except 26 who could not be located. Thirty-nine and five-tenths per cent were improved or much improved; 39.5 per cent were cured; 21 per cent were not improved. Some of them gradually growing worse, some of them remaining stationary. In this whole series of tonsillectomies there were no hopelessly chronic cases operated, and it would seem that the percentage of "not improved"—i. e., 21 per cent, should not have been so high; nevertheless, one feels that many of the cases that were marked "improved" would have become hopelessly chronic cases but for the tonsil surgery. A review of the cases of rheumatism marked "cured" is worth while. Of the 40 cases in which apparent cure resulted, 22 were diagnosed "chronic arthritis mild," 11 were diagnosed "chronic arthritis moderately severe," two were acute arthritis cases and five were diagnosed "myalgia." A general survey of these cases showed that most of these patients had suffered off and on for years, but not continuously, with pain and stiffness in one or several joints, at times acute and temporarily crippling. A few were crippled for months before operation. Orthopedic measures of various kinds had been tried. A few gave a history of symptoms of tonsil trouble; some no history of throat trouble. In seven cases the history showed an acute tonsillitis as the forerunner of the rheumatism. In other cases an accumulation in a tonsil crypt caused rheumatic symptoms. One such case bears reporting in detail.

Mr. H. A. S., male, age 32, seen first in 1909 on account of sore throat and cold in head. Tonsils were partly removed when a child. Attacks of sore throat without fever or disabling symptoms, three or four times a year. Antrum suppuration followed recent cold. Examination showed large red mass of tonsil tissue left side, small mass on right side. Advised tonsillectomy. Patient returned in 1910, 1911, 1913, 1914, 1915 on account of sore throat. At such times a large crypt in left tonsil was always found full of foul smelling material and tonsil tissue acutely inflamed. No quinsy, no fever. In April, 1915, patient remarked that aching during these attacks had become more pronounced and that his wrists would become so tender for a day or two that he could not grasp another's hand, and that after cleaning out the crypts, use of silver, gargle, etc., the wrist tenderness would rapidly disappear. His words were, "I'll be all right tomorrow." Six months later he submitted to a tonsillectomy. Five years later he reported he had never had a return of the rheumatism; had been absolutely relieved. This case is reported, not because of its rarity, but because it serves to bring up some points regarding the relations of a chronic tonsillar crypt infection to rheumatism.

It is the writer's observation that many adult patients who are cured or improved by tonsillectomy are of this type. It happened in this particular case that there was a direct connection noticed between the tonsil crypt inflammation and the rheumatism. In most cases no such connection is in evidence; yet the rapid and complete cure by tonsillectomy in such cases would lead one to believe that there is in some parts of the tonsil a similar diseased condition. A culture in this particular case showed streptococcus viridans and staphylococci. The question arises, what is the pathologic explanation of these rheumatic signs and symptoms that are so evanescent, lasting a day or two, only to recur, and finally to disappear altogether after removal of the tonsils? Surely there can be no secondary foci of bacteria in the joints, as in the persistent chronic infectious arthritic cases.

In a discussion on this question, the writer's attention was called to an explanation (by an internist who has not, I understand, published his viewpoint), that seems to the writer to be reasonable. This explanation is that at some time bacteria from the diseased tonsil tissue did reach the joint in question and caused a reaction in the tissues of the joint, likely the capsule, with the development of a sensitization of the joint. The bacteria in the joint lost their virulence and finally disintegrated. Subsequently during an acute exacerbation of the old chronic tonsil focus there occurs an absorption of toxin, without bacteria entering the blood stream, which produces an anaphylactic reaction in the joint that formerly was injured by bacterial localization. Pain and possibly temporary swelling of the joint or the capsule occurs and an acute rheumatic condition is in evidence, without fever and without permanency. It is common experience to see such rheumatic symptoms rapidly disappear. Whether these frequently recurring rheumatic signs and symptoms are caused by anaphylactic reactions or, on the contrary, by direct and frequent reinfection of the joint by bacteria is, of course, not susceptible of proof.

TABLE VIII.

Operations After Tonsillectomy	Cases
Abscessed teeth removed with improvement	8
Without improvement	13
With improvement—	
Colectomy	2
Appendectomy	2
Cholecystectomy	1
Nephrectomy	1
Tonsil stump	1
General Health	(Questionnaire Answers)
Improved	48
Much improved	30
Not changed	23
Unknown	29

In one case there was no improvement following tonsillectomy. On the contrary, some six or eight months afterwards patient developed an acute polyarthritis, endocarditis and nephritis. Our examination of the nose and throat failed to show any tonsil tissue or nasal sinusitis. The teeth had repeatedly been X-rayed with negative results. One tooth, however, was a dead one, and it was removed in the hope of helping the condition. There was considerable disease around the root of the tooth; culture showed streptococcus hemolyticus. This and a few other cases have shown to us the futility of relying absoluetly upon an X-ray examination of the teeth.

One of the two colectomy cases bears brief reporting.

E. M. C., age 35, referred January, 1916, because of frequent sore throats and rheumatism of five years' duration, dating back to attack of tonsillitis. Left ankle first affected, then both ankles, knees, wrists and fingers; swelling and severe pain at times. Constantly crippled during the past year. Has had treatment of various kinds, feels that diet has alone been of value. In February, 1915, fasted absolutely for seventeen days. After seven or eight days rheumatism rapidly decreased, almost disappeared, to return on beginning to eat. Rheumatism grew so severe that in June, 1915, fasted for 26 days, took nothing but water; weight dropped from 170 to 123 pounds, with almost complete relief from pain. Never any tooth trouble. Examination shows a large, strong, apparently healthy man, except for the polyarthritis; cannot raise his arm above level of shoulder nor close hands without pain; limps badly; can walk upstairs one step at a time, but painful. Heart and lungs normal; intestinal tract apparently normal; normal b. m. daily, no indigestion. Wassermann negative. Complement fixation test for streptococcus viridans negative. Complement fixation test for gonorrhea negative. Teeth sound, especially good; not a filling in any tooth. Tonsils show signs of marked chronic infection. Upper crypts contain cheesy masses; tonsils large, red and pulpy. This case is reported in some detail to show the history of effect of diet, the excellent general health, the perfect teeth, the definite initial tonsillitis history and the marked diseased appearance of the tonsils. In this case surely a tonsillectomy was indicated and good results expected. Except for a temporary improvement (due likely to decrease in diet during convalescence) the tonsillectomy was of no value. The rheumatism increased. A further study showed a colon dilatation and a nine-hr. stasis. A colectomy was finally done August, 1916, with marked and gradual improvement. Four and a half years later he reported much improved, working his ranch daily, some stiffness, no recurrence of severe pains; "85 per cent cured" (patient's estimate). It seems that the colectomy rather than the tonsillectomy was responsible for the improvement in this patient.

The figures shown in the general health table were obtained from questionnaires from the patients. It is rather noteworthy that 78 of the 111 patients reported their general health improved or much improved. Two cases died. Both were chronic cases in which organic heart disease had existed for years. They were operated on largely because of the heart condition rather than because of the rheumatism, which though existing was not severe. Both cases were operated on under local anesthetic, did well so far as the operation was concerned, but died two years after the operation without having received any value for the tonsillectomy. It was the opinion that the tonsillectomy had no ill effect on the heart.

CONCLUSIONS.

The difficulties of the problem of selection of operative cases are, of course, common to us all. It has been the writer's experience to find that adult patients suffering from so-called focal infection symptoms are referred to the laryngologist for his decision as to whether or not the tonsil is the seat of a chronic infection. Other patients come with the statement that their tonsils have been pronounced infected or that a culture that has been made from the crypts showed chronic infection. The writer has taken some pains to inform all such patients that every adult tonsil is a chronic infected tonsil, from which a positive culture can be made. The same is probably true of most tonsils in children. Therefore, the necessity for a tonsillectomy depends, not solely upon the examination by a laryngologist but upon a complete study of the patient to determine all possible factors responsible for the invalidism—rheumatism, heart trouble, etc. Charlatanism in the pseudospecialist has thrived to a large extent by virtue of the rather firmly rooted impression of the need of the removal of all infected tonsils. It seems to the writer that laryngologists should combat this idea, which is common, at least in my community, to a rather large part of the general medical profession and general public. Overenthusiasm on the part of many conscientious men has also been responsible for much unnecessary surgery. Sharp lines of selection of operative cases cannot, of course, be drawn, but there is urgent need at this time for careful group study of all tonsil patients, and a decision for or against tonsillectomy in any given case is ill advised until this has been done.

IX.

HYOSCIN AND MORPHIN AS A PRELIMINARY TO LOCAL ANESTHETICS.*

By Lee M. Hurd, M. D.,

New York.

In 1914, after using this method for nearly two years, I made a report.† Now, in the eighth year, stimulated by the findings of your committee on local anesthetics, reported before this section last year, I am prompted to take up this subject for a second time. Anything that adds to the comfort of the patient, lessens shock and reduces risk of local anesthetic intoxication is surely worth while.

In regard to the report of your committee on local anesthesia: the answers received to the question, "What toxic effects have come under your notice?" thirty-seven in number detail the symptoms of syncope or phrenic irritation. I consider that all were shock except two or three. The fatalities, excluding the mistakes in dosage, and those cases in which the anesthetic was injected into a vein, appear to me more like mental shock death, and not like fatality from local anesthesia.

The experiments on lower animals, except for toxic dosage, are of no help, because animals experience no apprehension or fear. In the human being, especially in the nervous type, which are in the great majority, apprehension and fear play a very important rôle in shock, even more important than the shock of operative trauma. Not only should the operative field be anesthetized, but the contact ceptors and the special sense ceptors should be blocked.

I wish to state again that the preliminary administration of scopolamin (hyoscin) hydrobromate and morphin sulphate has eliminated the unpleasant effects of local anesthesia, which I experienced in the past, and given me a confidence that, no matter how nervous or apprehensive patients may be, there will be no trouble with them at time of operation.

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*Read before the Section on Laryngology, Otology and Rhinology of the American Medical Association at the seventy-second annual session, held at Boston, Mass., June 6-10, 1921.

†Hurd, L. M.: Laryngoscope, November, 1914.

The operations of otolaryngology usually done under local anesthesia are expanded by scopolamin and morphin to the major group, such as mastoidectomies, laryngectomies and resections of the jaw, when general anesthesia is not desired.

DISADVANTAGE.

Though nearly all patients are ready forty-five minutes after receiving scopolamin and morphin, some require a longer time to settle down, and some require two or three doses. This makes it impossible to appoint a definite time for the operation, as is required in reserving the operating room in a busy hospital. I have had a delay of over two hours in some incidences.

The routine dose is morphin ¼ grain, scopolamin 1/100 grain. This is usually given after a light meal; if necessary, after forty-five minutes, I administer a second dose, usually half of the first dose, though this depends on whether the patient reacts to one drug more than to the other. If so, less of this is used in the second dose, and more of the less active drug.

In 300 consecutive cases a second dose was given in forty-two cases; a third dose in five cases, and a fourth dose in two cases. The third and fourth doses were usually given for long major operations. One of my first cases, a woman, became suddenly very hysterical when I was half way through the operation and was noisy, crying, but mainly laughing and noisy for several hours. In her case I had been forewarned of her hysteria; in the hospital, next day, she did not remember the hysterical outburst or any part of the operation. Four other women showed some hysteria when the first tonsil was removed, but gave no cause for complaint when the second came out. I had misjudged my patients and had not given them enough scopolamin and morphin. They probably would have been all right after the second dose.

Two of my early cases (which were mentioned in my previous report), while I was using the sitting position, had marked drop of blood pressure to 90 mg. systolic; otherwise their condition was good and the operation was finished. In the first case, I gave a hypodermic injection of 10 minims of epinephrin and placed him in a recumbent position. In the second case, after operation, the recumbent position promptly raised the blood pressure. For the last three years all cases have been kept recumbent during the operation, and, even if the dose is not sufficient, syncope and lowered blood pressure do not occur.

Previous to three years ago, the scopolamin I was using did not act as consistently as that which I am using now. There were more cases of delirium, coma, prolonged effect, etc. The scopolamin must be pure; especially, it must not contain apoatropin as an impurity. For the last three years the action of the scopolamin which I have used has been quite as consistent as the action of the morphin. The tablets must be kept dry and fresh solution made at the time of the operation, as scopolamin in solution will break down into impure and poisonous byproducts in from twenty-four to forty-eight hours.

The patients were nearly all good risks, though the poor surgical risks did as well. When patients are ready for operation, their pulse rate is reduced, they are sleepy or sleeping and do not want to be disturbed, speech is slow and somewhat thick, there is slowed cerebration, they may be somewhat confused, and in moving from bed to operating table they have a staggering gait. Pupils may be contracted (morphin) or dilated (scopolamin). The ideal is midway. The face usually itches, the color is good.

The pulse is taken at the time the patient enters the office for operation, when the operation begins, and immediately it is finished. The average pulse in fifty cases was 99 at the time of entering the office, 85 at the beginning of the operation, and 85 at the end of the operation. This does not, however, show the whole picture; for example, 128, 88, 84 in a highly nervous patient, and 76, 76, 72 in a phlegmatic one might be taken as extremes.

The blood pressure, which was taken in a few cases a day or two before, at the beginning of the operation and at the end, shows the variations indicated in Table 1.

TABLE 1. BLOOD PRESSURE.

Before Operation		Beginning of Oper'n		End of Operation	
Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
122	76	110	70	124	74
170	100	180	100	200	110
150	100	150	104	162	94
132	84	120	80	150	100

The age groups in 300 cases are given in Table 2. Scopolamin and morphin combined work well at all these ages.

TABLE 2. AGE GROUPS IN THREE HUNDRED CASES.

Age	No. of Cases	Percentage
From 11 to 20	50	16+
From 20 to 30	75	25
From 30 to 40	93	31
From 40 to 50	50	16+
From 50 to 60	18	6
From 60 to 70	14	5

Having had the usual run of syncopes and varying degrees of psychic shock before I began the use of scopolamin and morphin, I now have none with its use. The patient has a steady pulse, which tends to pass the anesthetic that is being absorbed on to the liver for elimination, preventing intoxication.

Morphin can be used in larger doses if cocain is used in conjunction, and the reverse is true. Scopolamin has the same physiologic action as atropin, plus blocking of the mind. Blocking the sensory nerves with morphin and scopolamin prevents shock, and though more local anesthesia can be used, as a matter of practice less anesthetic is necessary.

The local anesthetics used were: Cocain (topically), twenty-nine times; procain (hypodermically), forty-one times; alypin (topically), 223 times, and on two occasions no anesthetic was used.

Alypin and procain are quickly eliminated and are, therefore, better than cocain. Alypin, which is as anesthetic as cocain and much less toxic, I prefer, though it is unobtainable at present. Patients are kept in the office two or three hours after operation, during which time they are asleep. They are awakened then, and are usually in condition to be taken home. Since the previous report I have extended its use for all cases under local anesthesia, except the very minor and short operations, regardless of age or surgical risk. Whether the patient appears nervous or phlegmatic, I feel confident that if this method is used you will not have the symptoms attributed to anesthetic intoxication and the fatalities with the proper use of the local anesthetic will be greatly reduced.

39 East 50th Street.

X.

NASOCILIARY NEURALGIA.

By Greenfield Sluder, M. D.,

St. Louis.

Pain in the eyes, brow and root of the nose is frequent and may be of different origins. Localization of the pain of the several origins by the patient is not precise. Pains of frontal, ethmoidal or sphenoidal sinuses, or nasal (sphenopalatine-Meckel's) ganglion, or supraorbital or nasociliary nerve origin may overlap in their sensations. This last pain is usually referred to the small district bounded by the supraciliary ridge above the supraorbital notch laterally and the nasal bones below. Sometimes it extends to the tip of the nose. Rarely it is referred to the eyes. Patients often complain that they cannot wear their glasses because of soreness of the nasal bones. Severe degrees, however, of whatever origin, may be referred beyond these limits. Pain of supraorbital neuralgia is more widespread. Pain of suppuration of the frontal or ethmoidal sinuses, under pressure, is of long-known recognition. Pain of vacuum frontal sinus origin was described in 1900. Pain of nasal ganglion origin referred to this district was described in 1908. It is usually accompanied by other painful sensations. Pain of supraorbital systemic toxic neuralgia is of classical description.

Anatomic description, copied from Quain, eleventh edition, part II, iii, 16:

"Nasociliary or nasal nerve. This nerve enters the orbit between the heads of the external rectus muscle and between the two divisions of the third nerve. It then inclines inwards over the optic nerve, passing beneath the superior rectus and superior oblique muscles, to the inner side of the orbit, and leaves that cavity by the anterior internal orbital canal. In this part of its course it furnishes a slender branch to the ciliary ganglion, one or two filaments (long ciliary) directly to the eyeball, and a considerable infratrochlear branch, which arises just before the nerve enters its canal on the inner side of the orbit.

Arrived in the cranial cavity, the nerve is directed forwards in a groove at the outer edge of the cribriform plate of the ethmoid bone to a small canal between the forepart of the plate and the frontal bone, through which it descends to the nasal fossa. Here it gives off internal, or septal, and external branches to the mucous membrane of the forepart of the nasal fossa, and is then continued downwards in the groove on the back of the nasal bone, to terminate as the anterior or superficial branch in the integument of the lower part of the dorsum of the nose.

The branch to the ciliary ganglion, very slender and from a quarter to half an inch in length, arises generally between the heads of the external rectus. It lies on the outer side of the optic nerve and enters the upper and back part of the ganglion, constituting its long root.

The long ciliary nerves are situated on the inner side of the optic nerve; they join one or more of the short ciliary branches from the ciliary ganglion, and, after perforating the sclerotic coat of the eye, are distributed in the same manner as those nerves.

The infratrochlear nerve runs forwards along the inner side of the orbit, below the superior oblique muscle, and parallel to the supratrochlear nerve, from which it receives, near the pulley of the oblique muscle, a filament of connection. The nerve is then continued below the pulley to the inner angle of the eye, and ends in filaments which supply the conjunctiva, the caruncle and the lacrimal sac as well as the integument of the upper eyelid and root of the nose.

The internal or septal branch supplies the pituitary membrane over the forepart of the septum, extending downwards nearly as far as the opening of the nostril.

The external branch, often represented by two or three filaments, is distributed to the mucous membrane of the forepart of the outer wall of the nasal fossa, including the anterior ends of middle and lower turbinate bones.

The anterior or superficial branch issues between the nasal bone and the upper lateral nose and runs downwards under cover of the compressor naris muscle to the tip of the nose, supplying the skin of the lower part of the organ.

Varieties.—The nasociliary nerve occasionally (frequently, Krause) gives filaments to the superior and internal recti muscles. A branch to the lavator palpebræ superioris has also been met with (Fasebeck). In one case filaments of communication passed from a small ganglion connected with nasociliary nerve to the third and sixth nerves (Svitzer). In two instances Testut observed absence of the infratrochlear branch, its place being supplied by the supratrochlear nerve. Offsets from the nasociliary nerve, as it traverses the anterior internal orbital canal to the frontal sinus and ethmoid cells, are described by Meckel and Langenbeck; and a sphenoethmoidal (Luschka) or posterior ethmoidal (Krause) branch is said to pass through the posterior internal orbital canal to the mucous membrane of the sphenoid sinus and posterior ethmoid cells."

It is to be seen that the nasociliary nerve as it enters the nose in its uppermost anterior limit is quite near the surface of the membrane. It is more superficial than the nasal ganglion (usually). The ganglion may, however, be quite superficial (submucous), according to Krause. So it is easily understandable that an inflammation of the membrane in this part may irritate or inflame the nerve and produce pain. In older cases, with general hyperplasia of membrane and bone, it is also easily understandable that the ethmoid slit may be encroached upon to narrow its caliber and place the nerve in a more easily vulnerable position.

Differential Diagnosis.—The nasociliary nerve is more easily cocainized than the nasal ganglion. A small applicator with 20 per cent solution suffices. It is passed upward on the inside of the nose, being held forward in contact with the anterior limit of the nasal fossa until it reaches the roof of the fossa. By this procedure it arrives automatically in the apex of the angle formed by the cribriform plate above and the anterior limit of the nasal fossa. It is at this point that the nasociliary nerve enters the nasal fossa.

When the pain in this region is of nasociliary origin, such an application of cocain will stop it in a few minutes. Should it be of the other origins mentioned above, it will not be influenced by such an application. The differential diagnosis will, furthermore, reveal a tender spot in the floor of the frontal sinus if it be a case of vacuum or purulent frontal sinus origin (Ewing's sign). If it be a suppurating frontal or ethmoid sinus, pus will usually be seen in the middle meatus anteriorly. X-rays will show the frontal separated from the ethmoid. Should the frontal be totally closed, no pus will be thus seen; but the sinus will burst into the orbit. X-rays also show it. Should the pain be a supraorbital neuralgia, this nerve in the supraorbital notch will be found sensitive to touch. Pain of nasal ganglionic or sphenoidal origin will not be influenced by nasociliary cocainization. Ewing's sign will be absent. No pus will be found in the middle meatus. The supraorbital nerve will be negative to pressure. Pain of nasal ganglionic origin will be relieved by cocainization of the nasal ganglion. Pain of sphenoidal origin will not be influenced by any of the above procedures.

Clinical Course and Treatment.—My observation, so far, has been that nasociliary neuralgia has usually been a transitory phenomenon in the course of cases which have been under observation or treatment for something else. The nasociliary neuralgia would appear from time to time in the course of other clinical conditions and was usually not obstinate; it would yield to cocainization and applications of dilute carbolic acid, ½ per cent. Four times, however, I have seen it the only clinical phenomenon. Once I injected the nerve trunk at its entrance into the nose, with relief to the patient. The technic is that first advocated by Otto J. Stein in his alcohol injection treatment of hyperesthetic rhinitis. A straight needle is passed upward in the nose in the same way as the applicator for cocainization, described above. A few drops 95 per cent alcohol with 5 per cent phenol are instilled into the nerve trunk at its exit from the ethmoid slit.

XI.

VOCATIONAL, OCCUPATIONAL OR INDUSTRIAL DISEASES OF THE EAR, NOSE AND THROAT.

By John A. Donovan, M. D.,

Butte, Mont.

A correspondence with one or more otologists in each locality in North America shows that a few of our profession overemphasize this subject, while many more have hardly given it a thought. The recent war, as well as the increasing clamor for state medicine, makes it imperative that we consider it carefully. A general knowledge will also assist us in making a correct diagnosis in many an obscure case, especially in patients consulting us from other industrial localities. Besides one or two friends in each city, who have assisted me, to whom I express thanks, I have quoted liberally from H. V. Wurdemann, also from Kober-Hanson, and Barrett and Shaw's works on "Occupational and Vocational Diseases."

Though it may appear that all the diseases enumerated here are due to a vocation, such is far from the intention, for, except in those few very obvious special cases, the vast majority would not have resulted except for a very fertile soil being already supplied, such as diseased tonsils, adenoids, deflected septum, hypertrophied turbinates, or other abnormalities of tissue. Any of these conditions should always immediately receive special attention when the person contemplates a vocation which is likely to aggravate the condition, though no serious symptoms then manifest.

Not knowing a better method, I shall ask your kind indulgence, first, in considering general conditions and specific elements causing diseases, together with the symptoms of each. Then, under vocations, I will simply refer to these causes again to save your time. With few exceptions, no symptoms, except those to which this title refers, will be mentioned, the other general symptoms being assumed.

CAUSES.

1. Altitude.—In first visiting high altitudes most people experience a fullness in the head, a feeling of dyspnea, head throbbing with heartbeats, nose bleed, nasal turgescence, irritation and dryness of the throat, fullness (really a vacuum) in the ears, tinnitus, decreased hearing (really due to the closure of the tubes, and immediately relieved by inflation), and dryness of all mucous membranes. High altitudes are naturally very dry. Exercise, especially climbing, increases all symptoms. All these usually disappear in from a few hours to a few weeks' sojourn. A very rapid ascent to a high altitude intensifies all these symptoms. Besides there may be severe hemorrhage from the nose, gums and even the ears, with labyrinth and semicircular canal involvement, dizziness, nausea and even complete unconsciousness. This latter condition is more likely produced by a too rapid descent, and results fatally for the aviator (the cause of a number of fatalities).

Of course, cloud moisture, alternating with extreme dryness and intense cold, accompany extreme elevations. Fortunately people soon become accustomed to these minor changes and do not notice them. For example, in Butte, where thousands of men descend and ascend 3,000 feet, or even 4,000 feet, in the mines in a few minutes' time, I do not recall any miner ever mentioning this factor, while a stranger is fully conscious of a change. Of course the heat would be great at these lower levels, except for the constant supply of compressed air.

Caisson disease is in this classification. Men work in highly compressed air containers. Probably there is an excess of oxygen, and possibly injurious gases, dissolved under pressure in the blood; and if the air is allowed to escape too rapidly from the caisson this gas in the blood is released into the tissues; or it may be toxic or carbon dioxid; or again it may simply be from the sudden release of pressure. The results are: Epistaxis (very common), ruptured drum membranes, tinnitus, nausea, giddiness, loss of power in the limbs and unconsciousness.

Asthma, hay fever and similar conditions are usually relieved and very frequently permanently disappear in high, dry altitudes. Hypertrophies, turgescence, tinnitus, etc., are probably made worse, but often the dryness improves them. Mucous discharges are lessened. Inversely, these chronic turgescent rhinitis, with ear complications, or dry or atrophic conditions, improve immediately on going to low, moist altitudes. I have had many patients with chronic discharging sinuses or ears report immediate relief on going lower, while a very few became worse by the change. We are about 5,800 feet elevation, dry, and never very hot. Contrary to the general opinion, a descending barometer is not the cause, in itself, of rheumatic conditions, as otherwise elevation would increase it also, which is not the case.

2. Winds and Drafts.—Fast motorists, all speed fiends and those living or working in high winds or drafts suffer from irritation of the nose and throat, with all the ear complications. A direct hard wind into the ear causes myringitis, otitis and, in time, chronic disturbances with deafness.

3. Temperature.—Constant extreme cold or heat do not affect like sudden changes. The extreme cold of the North Pole did not give Peary's men any colds, but opening a bundle of carpets, also cleaning house, immediately produced severe coryza in all hands. The modern girl, or rather her modern clothing, or lack of it, will not produce nearly the coryzas, rhinitis, laryngitis and ear complications from which her more or less fortunate sisters cooped up in overheated apartments will suffer. The overheated offices, theaters and Pullman cars in which the long suffering public is at the mercy of a janitor or colored porter cause innumerable catarrhal conditions and are the origin of too many fatalities resulting therefrom. It is the excessive heat, relieved by sudden cold drafts, or vice versa, that causes the trouble. Of course, local irritations, dermitis, perichondritis and effusions in the external ear, frost bites and gangrene of the ear result from extreme exposures.

Many a case of chronic laryngitis will soon disappear by sojourning outdoors. Living in a tent in the woods in winter is my favorite prescription for myself.

4. Humidity.—Humidity, according to Collis and Greenwood. Though in dry, changing air one may work at a temperature of 135 degrees F. without any immediate harmful

results, in humidity, when the temperature as measured by the wet bulb thermometer reaches 78 degrees F., the danger point is reached for carrying on active work. Body temperature is regulated principally by skin radiation, conduction and evaporation, which is prevented by a warm humid atmosphere. The maximum efficiency seems to be obtained in a relatively dry atmosphere at 65 degrees F., while after a day's labor inside, a humid temperature of 70 degrees F., the worker has no energy left and has no desire for food.

In dry places, mucous membranes dry and discharges diminish or cease. In dampness, mucous discharges become excessive. Rheumatism, and its sore throats, is common. Pharyngitis, tonsillitis and laryngitis are common in those working in wet places, also mycosis of the external ear canal, chronic congestion of all mucous membranes (these, of course, are aggravated by the sudden cooling at the close of day).

5. Dust.—Dust, except when fully exposed to sunlight and fresh air, usually contains infectious matter which is the cause of a great many of the infectious diseases of the respiratory organs, with the usual ear complications. Mechanically, there is a constant irritation of all the mucous membranes. The anterior part of the septum is irritated and bleeds easily, and there are small ulcerations and perforations. The posterior septum is swollen and dust covered. Ultimately there are hypertrophies of the posterior, especially the upper portion of the septum, the middle, and then the lower turbinates, functional collapse of the nasal tissue, loss of smell, taste, etc., dry feeling in the throat, engorgement of the large vessels, dusty mucous, glaring pharynx and many small hypertrophies, later loss of sensation and atrophy. The larynx shows similar reaction, ear complications as usual.

Most varieties of dust, ordinary dirt, smoke, coal dust, etc., will be coughed up and expectorated, so, if not exposed too long, will give but temporary symptoms. Every person with a deflected septum is an easy victim to dust irritation. Hard rock dust, included under the general term silica, remains permanently in the lungs, producing a fibrous condition known as fibroid phthisis, or silicosis, or pneumonoconiosis, in popular parlance "miners' con." With the exception of the constant use of water sprayed on the rock while working, nothing will prevent this most serious malady. The underground miner cannot wear a mask at all comfortably, but the man outdoors might. In fact, it is at present his only salvation. Butte's 3,000 miles of underground tunnels are kept habitable and cooled by an abundant supply of fresh air and cold water, operating through the compressed air drills. Fresh air is blown in the mine through large canvas tubes. The former method—that is, the use of compressed air—carried too much moisture. Of course, there is still some dust, and considerable heat and moisture, so we do not recommend deep mines as an ideal health resort. Though in some mines chronic laryngitis is common, in ours it is extremely rare proportionately.

Dust causes hay fever and asthma in many, especially when loaded with organic matter from sweepings, such as flour, horse dust and the pollen of many plants, etc., but again, there is most likely to be a deflected septum and enlarged turbinates, adenoids or tonsils, making an ideal field for trouble.

Ankylostomiasis, in miners, does not appear to exist in America. It is a trematode worm disease with anemia and edema.

6. Chemical Irritants.—Arsenic dust is a very frequent cause of a low anterior septum perforation and often occurs without noticeable symptoms. It does not extend to the bony septum or high up. Aside from this, acne of the face, nose and ears (arsenic polk), boils, ulcers, nausea, vomiting, severe coryza, dry sore throat and hoarseness frequently result from living in rooms with arsenic colored wallpaper. This arsenic dust appears to result from bacterial action originating in the adhesive paste. Middle ear and auditory nerve affections may occur.

Aniline. Local ulcers of the skin and mucous membranes, with dermatitis.

Carbon. Chimney dust, smoke, tar, pitch, etc., are local irritants, coryza and not infrequently cancer resulting.

Carbon bisulphid. Peripheral neuritis, bilateral paralysis.

Carbon monoxid, dioxid. Diminished power of speech and hearing, dizziness, extreme headache, aggravated by exertion.

Chrome and bichromates. Eczema and erosions, skin ulcers with thick undermined edges. It attacks nasal septum similar to arsenic, rapid perforation.

Cement, lime, salt, soda and potash are very irritating to the septum, causing small ulcers, hemorrhages and perforations.

Gasoline, benzol and naphtha cause irritation, with slow necrosis, etc.

Iodin is a nose and throat irritant, causing tinnitus and double hearing.

Fumes and gases of acids, alkalies and many other substances are extremely irritating to all mucous membranes, many very toxic, with symptoms accordingly.

Lead. Blue line on gums, pale face, headaches, simulating brain tumor, tongue and lip tremors, interrupted speech, laryngeal muscle paralysis, tinnitus, auditory paralysis and double wrist drop. Lead poisoning occurs in upwards of 110 industries.

Manganese. Impulsive laughter, mental changes, monotonous voice.

Mercury. Pale face, sore painful mouth, bleeding gums, profuse salivation, difficult swallowing, yellow ulcers, foul odors; in chronic cases a peculiar copper color in the mouth and trachea, constriction of the esophagus, labyrinth deafness, auditory paralysis and necrosis of the bone.

Phosphorus. Skin pallor, intense pain in the lower gums, necrosis of the lower jaw (phosyjaw), pus, large glands, necrosis of the septum and possibly the ear bones also.

Platinum dust. Coryza, sneezing, hay fever from which those exposed can get no relief while vocation continues.

Zinc. Quite similar to arsenic.

7. Explosives.—In a paper, "Blasting Eye Injuries," read before the Section of Ophthalmology, A. M. A., 1905, Journal A. M. A., I pointed out that gunpowder has a rendering effect, while high explosives have a shattering effect. Much surprise has been expressed at the numerous ear troubles following the late war, when none occurred in earlier wars. The substitution for black powder of nitroglycerin, fulminates, trinitrate of tulol, etc., is the explanation. Though a detached retina but very rarely, if ever, occurs from gunpowder explosion and not frequently from dynamite, when a box of fulminate detonating caps explodes accidentally, by observation of the patients who were in close proximity, that detach-

ment is the rule. Thus by analogy the severe ear complications are readily understood.

A shot from an ordinary sporting rifle, gun or pistol, if near the head, produces a concussion in the ears, irritation and tinnitus. I have found that hot douches of considerable water will readily relieve this. The ears should be protected with paraffined cotton plugs or something similar. If the concussion from big guns or blasts is severe and the membrane ruptures, it follows the long head of the malleus. If infection follows, then suppurative otitis results, with its usual complications. If the shock is severe and rupture fails to occur, the shock is transmitted to the internal ear, with possibly small labyrinth hemorrhages, symptoms of tinnitus, and decreased or lost hearing. The high and low scales are markedly diminished, while the middle scale may remain normal. Fortunately, most cases practically recover. When they do not, degeneration takes place in the organ of Corti and the labyrinth, with possibly eighth nerve involvement. Of course, severe shell shock, a process complex, may produce central trouble causing deafness. The semicircular canals are involved, with loss of equilibrium, dizziness, etc. Hysteria may be the cause with a certain class of patients. Malingering is not under discussion here. With riflemen and shooters in general, a mild catarrhal otitis media is much aggravated—in fact, it is a forerunner of many of the symptoms brought on by exposure to these blasts. Nitroglycerin headaches result from its explosion in confined spaces in mines, etc.

8. Noise.—Here we differ widely, and your criticism is especially invited. To economize time, I shall be dogmatic and simply express my own conclusions after studying many diverse opinions. The constant irritating noises that drive one mad belong to the field of the neurologist, but do, of course, consume much valuable mental energy. Noises of high pitch, when constant, especially within small inclosures, or within close proximity, do produce deafness. This is transmitted directly through the sound conducting mechanism, and not otherwise, and can be prevented by protecting the ears directly from the noises. There is a destruction of the organ of Corti in severe cases, beginning in the nucleated cells, to the neuron, then to the vibrating mechanism and membranous labyrinth.

The canals may become involved, vertigo resulting. Within buildings hair felt covers and felt partitions reduce the noise very much. Something should be worn in the external ear canal also. Here again those afflicted with mild catarrhal tubes are the first to suffer.

I believe that deafness from explosions is due directly to concussion and not to noises.

9. Fatigue.—Fatigue lessens tonicity and resistance of the mucous membranes in the nose and throat, with resulting engorgement. It also decreases the power of smell, speech and hearing. Disinclination to work, or aversion to work, brings on fatigue much faster than real labor. Poor hygienic conditions, bad light, bad air and most of the aforementioned abnormal conditions produce fatigue more rapidly than well ventilated, well lighted surroundings. A vacation or temporary change of vocation lessens fatigue and produces general relaxation from strain and is a direct invigorator. In other words, take a vacation out of doors while yet you may, for the time comes soon when you will not be able to say where you will go next. Join the Boy Scouts on their camping trips.

10. Vocations.—Alcoholics, a common vocation of the past ages. Engorged nasal and pharyngeal mucous membranes, which in time become chronic. All abnormalities of the nose, throat and ears are made worse. A characteristic unsteady voice.

Authors. See office workers.

Auto drivers, exposed to drafts, nose, throat and ears, dust and gas, with the advantage of much fresh air.

Attorneys. See singers and office workers.

Aniline dye workers. Dermatitis and ulcers.

Aviators. See altitude. In addition they have exposure to high winds and cold, also to the intense constant motor noises, with resulting deafness if not protected.

Athletes. Septum hematoma, broken septum, ruptured drums, perichondritis (prize fighter's ear).

Artificial flower makers. See arsenic and office workers.

Band instrument makers. See soldering.

Balloonists. See aviators.

Boilermakers' deafness. See noises.

Blacksmiths with deafness, very rare, except in close inclosures or where many are working together.

Brass founders' ague, headaches. See zinc.

Brass and copper workers. Green lime on gums. See zinc and noises.

Button, horn and shell workers. Dust very heavy, nose bleed, etc.

Brewers. Tuberculosis. See alcohol and humidity.

Blanket storing. Brimstone fumes very irritating.

Blasting. Powder headaches, deafness. See explosions.

Battery makers. See lead.

Bakers, confectioners, pastry cooks. Of their total deaths, one-third are due to diseases of the air passages and lungs. See dust and temperature.

Chauffeurs. See auto and carbon monoxid gas.

Cotton mills. See textile.

Coppersmiths. See noises and brass.

Cooks. Atrophy of taste and smell. See bakers.

Clergymen. Sore throats. See singers and office.

Color factory workers. See arsenic, soda, potash under cement.

Chimney sweeps. See cardon dust, cancer.

Canning factories. See humidity, wet and acid fumes from soldering.

Celluloid makers. Fumes from solvents, acetone, amylacetate, alcohol, ether and acids.

Chemists. See all chemical poisons and fumes, also office.

Cement and concrete workers. See cement and dust.

Chrome workers. See chrome poisoning.

Caisson workers. See altitude and humidity.

Divers and swimmers. Rupture of the tympanic membrane, acute infection of the middle ear from water entering the middle ear through the eustachian tubes, due to improper breathing.

Dyers. Soda, potash. See calcium, aniline, lead and arsenic.

Dentists. See office workers.

Electric generators. Deafness from noise, when cooling motors improperly installed.

Elevator boys. See temperature and drafts.

Explosive factory workers. Powder (nitroglycerin) headaches. See carbon monoxid and celluloid makers.

Engineers. See locomotive.

Farmers. See dust, temperature and humidity. See stockmen, tetanus, foreign bodies, foxtail, chaff and burrs in the external ear canal often remain a long time before they are noticed.

Fur pellers. Anthrax.

Flax dressing. Silicosis. See dust.

Flour millers. See dust and noises. (Millers' asthma.)

Fishermen. Dampness and cold. See temperature and humidity. Case of lymphoid growth in the tongue and epiglottis reported by Dr. C. H. Baker.

Fireworks makers. Argyria from breathing silver dust.

Garage workers. See chauffeurs.

Gas works. Carbon monoxid poisoning, and dust.

Glass blowers' mouth. Swelling from the angle of the mouth to below the ears and parotid glands. Thick patches of mucous membrane on the cheeks, tuberculosis from excessive heat. Syphilis from exchange of blowing tools.

Dictaphone. Hal Foster reports deafness. See telephone.

Gold milling. See dust, fumes, chlorin, cyanid and mercury.

Galvanizing. See arsenic, also mercury and zinc.

Gannister miners and workers. See stone cutters.

Hat felt makers. See mercury.

Horsemen. See stockmen, also athletes.

Indoor workers. See dust and noises.

Janitors. Hay fever. See dust.

Jute factory workers. Tetanus.

Lime workers, plasterers, etc. See cement.

Lead products manufacturing. See lead.

Leather workers. See tanners.

Lawyers. See office and singers.

Locomotive engineers. These have heretofore been erroneously placed with boilermakers. All authors and most of my correspondents so classified them. For many years I have taken care of all Northern Pacific employes operating trains across the Rocky Mountains; also the employes of the B. A. & P. R. R., the first electrified steam railroad. The mountain division is the hardest and most noisy portion of any railroad;

even rear coach passengers can hear the puffing locomotives, yet I have never treated a trainman for deafness that I could positively directly attribute either to the noise or vibrations.

President H. B. Smith and Secretary W. A. Laidlaw of the Northern Pacific Beneficiary Association report to me that a hurried search through several years' records confirms my observations. Through the courtesy of Mr. F. V. Whiting, attorney of their claim department, I was put in communication with the chief surgeons of the New York Central systems and the results were practically the same. A German authority reports that only 8 per cent of their locomotive engineers have normal hearing. If this is correct, then the protection given to their engine drivers must be of the medieval type. Trainmen on modern equipped American roads are of course somewhat, though not excessively, exposed to conditions producing catarrh but not to noise or concussion deafness. I am excluding boiler factories, of course. I have seen one case of deafness in an engineer who was very close to the whistle when someone pulled it; also one case of deafness from a boiler explosion.

Match makers. See phosphorus.

Match safety. See chromates and phosphorus.

Mirror makers. See mercury.

Manufacturers of scientific instruments. See mercury and chemicals.

Mechanics. See locomotive engineers, noises and dust.

Millers. See dust, noise and millers' asthma.

Miners. See dust, humidity, temperature, altitude and explosions. Job, chapter 28, is the first miners' textbook written.

Miners' nystagmus is not an ear, but an eye complication.

Mountaineers. See altitude. The high, dry air, filled with ozone and a superabundance of ultraviolet rays, stimulating and invigorating, is the salvation of thousands of catarrhal, emaciated, overworked people who can occasionally avail themselves of its advantages.

Musical instrument makers. See makers of band instruments.

Office and indoor workers. Professional, clerical, clerks, factory girls, needleworkers, seamstresses. I believe this class supplies the head specialist his greatest number of patients, notwithstanding the fact that the authors have given it scant attention. G. W. Spohn in his letter feared he would be considered freakish for placing this occupation as the most hazardous in this special classification. I agree with him. A lack of outdoor physical exercise, poor air, overheated drafty rooms frequently filled with tobacco smoke, inappropriate diet, an excess of sweets and pastry, poor digestion and inactive bowels are all extremely conducive to edematous, sluggish mucous membranes, with a lack of tonicity, insufficient muscular strength in the pharynx, larynx and even within the ears. Catarrhal affections of the nose, throat and ears are the rule rather than the exception. When occasionally exposed to unusual atmospheric conditions, acute catarrhal inflammations immediately result, with tuberculosis, etc., following. Early appropriate treatment of nasal deformities, adenoids, tonsils and teeth will prevent the vast majority of these conditions.

Printers. Auditory nerve deafness, tuberculosis. See lead and vapors.

Plasterers. See cement.

Public speakers. See singers and office.

Pregnancy with some is accompanied by irritation and turgescence of the tuberculs of the septum (the genital area in the nose), paresthesia, and hyperesthesia of voice, smell and hearing.

Prize fighters' ears. See athletes.

Painters. See lead, arsenic and zinc.

Potters. See dust, silicosis and lead.

Physicians. See office. Liable to acute and chronic infections of the nose and throat, with sinus disease. Obstetricians, urologists and laryngologists exposed to syphilitic and tubercular infections, and Vincent's angina.

Power plants. See electric generators.

Porcelain workers. See chrome and lead.

Photographers. See office, soda, potash and chromes. Platinum dust from papers causes coryza and hay fever, sometimes very serious.

Powder makers. See explosions, gases and dust.

Rubber manufacturers. See carbon bisulphid and lead.

Rag grinders. See dust. Shoddy fever, severe irritation of all mucous membranes, with headaches.

Refrigerator and ice cream works. Chronic congestion (cold and moisture).

Syphilis. It is a disease affecting all vocations and avocations, but possibly more prevalent with those who are in close contact with the larger mass of the traveling public, society satellites, indoor sports, certain classes of rooming house, hotel and restaurant employes, physicians, especially obstetricians and urologists, nurses and wet nurses. My brief observation of the northern Indians was that they are extremely susceptible—in fact, it is very fatal amongst their younger women. Our present government regulations are a great help and, when efficiently administered, may ultimately eliminate it. Contrary to the prevalent opinion, the laryngologist sees the most cases; especially is this so with women patients.

Acute pharyngitis, chronic laryngitis, soft, soggy nasal growths easily broken down, perforations destroying the bone, with depression of the nose (not so with dust or chemical ulcers), laryngeal growths, loss of voice, deafness, cerebral troubles. Deep, dirty grayish diphtheritic appearing ulcers of pharynx, tonsils, palate and larynx, emitting a dirty dog odor, very characteristic, and when learned is almost a positive diagnostic symptom.

Stockmen. See farmers. Glanders, actinomycosis, anthrax, tetanus, foot and mouth disease ("aphtha fever"), vesicles and ulcers on the tongue, lips and pharynx (a long list of catalogued diseases officially designated, which very rarely happen), but foxtail beards (something like barley), burrs, etc., carried by the wind, frequently lodge in the ear canals, and sooner or later inflammation sets in. Though very frequently exposed to much wet, cold and severe high winds, etc. (excepting those with marked nasopharyngeal abnormalities and diseased teeth), they are singularly free of disease. A good diet of a reasonable allowance of that most nutritious, easily digested, stimulating food, good beef with vegetables, seems to build an immunity against the diseases we are here considering.

Seamstresses, needleworkers and dressmakers. See office. They seem especially prone to deafness.

Sheep tenders. See stockmen, and arsenic from sheep dip.

Soldiers. See explosives and syphilis.

Sailors. Mal de mer, an abnormal condition of the superior semicircular canals. See syphilis.

Slaughter house. Dampness, glanders, tetanus, actinomycosis, rheumatic throat, etc.

Subway workers. Carbon dioxid, iron dust, silicosis, drafts, temperature and moisture.

Swimmers. See divers.

Soldering. Fumes of acid produce intense pharyngitis, not infrequently becoming very serious.

Shoemaking and staining. Dust, aniline, lead, tuberculosis.

Sugar factories. The lymphatics from microorganisms, bone black dust, offensive fumes, catarrh.

Salt workers. See calcium.

Smeltermen. Arsenic, chlorine, bichromates, mercury, dust and chemicals.

Stone cutters. First in the list of vocations in percentage of tuberculosis mortality; silicosis, chronic diseases of all air passages and ears, followed by atrophy and laryngitis, etc.

Singers and speakers. Singers' cramp from fatigue in attempting to use the voice at too high a pitch or too great a range. Singing should not be done over one hour (two hours at the most) a day, and never over fifteen minutes (twenty minutes the extreme) at one time. There should be at least six weeks' vacation each year with no practice. Walking outdoors several miles a day is an ideal exercise and is absolutely necessary. Coughing to clear the throat when starting to sing induces hoarseness. Wear light woolen underwear in cold weather, no neck wrappings. No smoke, no dust.

Vocal nodules from excess or misuse of the voice. Laryngeal irritation affects the timbre of the voice. Too rapid training affects solidity. Vocal excesses diminish the compass. An emaciated condition, tuberculosis, etc., reduces the agility, also the intensity. Chest disorders reduce the medium scale. Catarrhal conditions, bad tonsils and adenoids diminish the clearness. Nasal abnormalities diminish the resonance. Neuropathic temperaments produce the nervous voice.

Speech fatigues more than singing. Public speakers should acquire and maintain a standard pitch, within the medium tones, which does not produce fatigue. Start speaking with the voice where it is and allow it to gradually reach its proper acquired standard intensity. Chronic pharyngitis and clergymen's throat, also laryngitis, results from the neglect of the above rules. See office.

Trainmen. See locomotives and drafts.

Tunnels. See miners, caisson and subways.

Travelers. See heat, Pullman and syphilis.

Theaters. See heat and atmosphere.

Tea, tobacco, spice and perfume testers. Nicotine, thein poisoning and atrophy.

Tobacco. Tobacco factory workers rank second in tubercular deaths from tobacco dust in the throat. Smoke aggravates catarrhal conditions and tinnitus is intensified. Cancer of the lip, syphilis from exchanging pipes.

Teachers. See office. Much catarrhal inflammation, chalk dust, irregular temperature of rooms, loud talking, laryngitis and secondary ear diseases.

Telephone. Operators in the Bell and all other phones of low voltage appear to have no more ear trouble than any other office workers (contrary to all textbook authorities). High tension currents, acoustic bangs, ringing bell with the receiver in the ear, especially so in the higher voltage systems, does cause annoyance, and does irritate a sensitive ear considerably, but ordinary conversation does not. I saw one man who lost the hearing in one ear completely by lightning striking a wire. Proper fusing would have prevented this. Dr. C. B. Lyman and Dr. E. W. Collins of the Rocky Mountain Bell System report no serious trouble at all with any operators, but report complaints to be in direct proportion to the user's mental attitude toward the company.

Tanning, leather workers, taxidermists and furriers. Asthma, anthrax, chrome, lead, arsenic, mercury and dust.

Textile workers. See dust and noises. Dry throats, cereum plugs in the ears, mycosis of the ear canals.

Upholsterers and mattress workers. Erysipelas and infections from old rags and dust. See textile.

Wool sorters' disease. See anthrax and mycosis of the ear caanls.

Wallpaper. See arsenic

Wood polishers. See chrome and dust.

Wheel polishers and grinders. See silicosis. Stone cutters.

Washerwomen. Infections, syphilis, chemicals (lead and mercury) from clothing of workers. Catarrhal conditions from temperature and moisture, mycosis of the ear canals.

Weavers and spinners. Mycosis of the ear canals.

A very formidable list, notwithstanding the Irishman's conclusions that, after all, nearly everybody dies in bed.

XII.

TINNITUS AURIUM.*

By Thos. J. Harris, M. D.,

New York.

Allow me to express to you my grateful appreciation for the invitation to address you this evening. It is with no little hesitation, however, that I venture to present before so distinguished a body of specialists a paper on the subject which has been assigned to me.

Boston's contributions to otologic science, from the days of the late lamented Dr. Clarence Blake, are exceeded in number and quality by no city in this country. To contribute anything new on the subject of tinnitus aurium is beyond my fondest hope. All subjects, however, connected with our specialty are deserving of consideration from time to time, and it is with the thought that I shall only be opening the discussion that I have the temerity to draw attention this evening to some of the salient features of tinnitus aurium.

Some twenty years ago the writer had the privilege of reading a short paper on this subject before one of our national societies, in which an attempt was made to bring together the not very extensive literature. Since that time, apart from a number of interesting case reports, only three articles of importance, so far as we are acquainted, have appeared in English, one in 1904, by W. Sohier Bryant, which contains the most elaborate classification of the subject which we know of, and the others by H. O. Reik, in 1904 and 1912, both of which represent much careful and meritorious study.

Tinnitus aurium (noises in the ear), for the most part a purely subjective sensation, intangible and invisible, must always remain a phenomenon, impossible in many particulars of complete solution. For its intelligent consideration, some simple working classification is desirable. None is simpler than a division into two groups, subjective and objective, according to whether the sound is heard only by the patient or by the observer as well, and again into two subdivisions, exaural and entotic, depending upon whether the sound arises outside or inside the ear. No symptom or condition arising in connection with the ear demands more careful investigation. A perfunctory examination is too often the practice. A faulty classification and prognosis is the result, with faliure in the majority of cases in the attempt at relief.

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*Read before the New England Ear, Nose and Throat Society, March 12, 1921.

Tinnitus aurium is wont to be found in the course of almost every affection of the ear, although it is true that some of the most severe cases of disease of the inner ear have been free from it. It is especially common as an early symptom of otosclerosis, often before any loss of hearing can be detected. At the onset its location by the patient is vague or faulty, but in time he becomes able to state with definiteness the locality from which it arises. The character of the noise varies in the extreme. Often, it is not one noise, but a number of noises associated together. The intensity of the noise in the two ears is apt to vary, and conditions of the weather, dampness, etc., bodily and mental exertion, alcohol, menstruation, etc., are wont to have a marked effect. The disposition of the patient also has much to do with the annoyance which the noise occasions. Some patients suffer so that they threaten to take their lives, while others apparently are little inconvenienced by it.

Cases of objective tinnitus are comparatively rare. The sounds may arise outside the ear and be loud enough to be heard at a distance. All of you have probably had cases of this nature. They may be vascular in character, arising from one of the vessels in the neck as demonstrated by digital compression, or muscular, due to the contraction of some one of the pharyngeal muscles. At times it is entotic, as for example, the sticky mucous sound which is occasioned by the physiologic act of opening the eustachian tube or the contraction of the tensor tympanic or stapedius muscles. Cases have been reported where this muscle contraction was voluntary.

Another group is tinnitus from reflex causes, as for example, from the trigeminal or facial nerve. Politzer speaks of a patient in whom rubbing the skin in the region of the ear would produce such a tinnitus. All of these noises are, for the most part, what we might call curiosities and do not demand lengthy consideration. The great majority of ear noises are subjective. Of these a few are exaural, as from an aneurism or large vessel in the neck, and of these fully 80 per cent are entotic. These can arise in connection with and as a symptom, at some stage or other, of almost every form of disease affecting the sound conducting or sound receiving apparatus, and apparently, at times, independent of all disease. A noted otologist in this country has had tinnitus for many years without deafness. Apart from the actual disease in the ear itself, and as a direct cause producing the ear trouble, is some obstructive disease of the nose and throat. Too great emphasis cannot be placed on the close relation between disease of the nose and throat and tinnitus aurium. Only second in importance as an etiologic factor is some auto-infection proceeding from the gastrointestinal tract. A number of cases have been reported where the source of the infection was in the teeth. Blood dyscrasias, such as lues, high pressure, pyemia, are also a cause. Certain drugs, such as quinin, salicylic acid, as you are all aware,can give rise to noises in the ear.

Still another group is the cases of nervous tinnitus without deafness, occurring in highly nervous individuals, due to anemia, grief, etc. If the cause is not removed the tinnitus may continue through life, usually without the onset of deafness.

Finally, in this connection, should be mentioned the interesting group of cases which Bryant among others has called attention to, namely, the psychic group. Here the complaint is of hearing voices. Patients are either already insane or the hallucinations complained of are premonitory symptoms of insanity, although Politzer states that where ear disease is present treatment addressed to it can improve the hallucinations or even cause them to disappear.

Bryant's conclusions, in his interesting paper on the subject of tinnitus aurium and hallucinations of hearing, bear out this contention of Politzer. He states: "The hallucinations usually depend for their inception on stimuli received by the auditory center. The stimuli originating peripherally pass directly along the auditory fibers or indirectly from other centers along the association tracts. In rare cases the auditory center itself may be subject to primary stimulation, which is due to pressure or to chemical irritants." He adds that "tinnitus aurium is a common accompaniment of auditory hallucination and is probably its usual exciting cause," and states "that the insane whom he had examined had chronic ear affection, which in all cases of recent insanity must have preceded the hallucinations of hearing. He concludes his article as follows:

"The evidence points out a logical connection between ear disease and hallucinations of hearing.

"In a susceptible, psychopathic individual, hallucinations may be excited by the irritation of subjective noises.

"Improvement or cure of the coincident ear affection may logically be expected to cause an improvement or cure of the auditory hallucination."

While Bryant's article is the most exhaustive on the subject with which we are acquainted, the relation between the ear disease and the presence of noises of the ear of the insane has long been known. The most famous instance of this is the musician Schumann, who, for a number of years before he was adjudged totally insane, used to hear strange voices. On one occasion he stated that he had heard the voices of Schubert and Mendelssohn, and as a result of this composed a selection in nine variations.

Just how subjective tinnitus aurium arises is not clearly understood. The usual view held is that it is due to increased labyrinthine pressure. This is satisfactory so far as the small number of cases having their origin in the inner ear is concerned. There is a certain number, as just stated, in which it is undoubtedly circulatory, transmitted from the great vessels of the neck, especially the carotid and the vertebral arteries, as shown by compressing these arteries. The larger group, however, is made up of cases of interference with the sound conducting apparatus. It is undoubtedly true that anything which serves to interfere with the escape of sound from the ear must needs increase it in intensity and so render it liable to be heard. Indeed one of the many wonders of the auditory apparatus is the marvelous way in which all entotic sounds are taken care of in health by the nervous system.

As opposed to the increased labyrinthine pressure theory is the view of Reik. Reik, as the result of a series of interesting experiments on dogs, showed that increase in the intratympanic pressure caused a vasomotor dilatation, resulting in a lowering of the blood pressure. Based on these experiments, it is Reik's opinion that "Subjective auditory sensations generally result from vasodilatory changes in the vasomotor systems of the middle or internal ear, and may be caused by any case of irritation which will so affect the vasomotor nerves controlling these vessels. Consequently, that stimulation of vasomotor of the ear produces a depressor effect, small vessels become dilated and the abnormally large amount of blood coursing through this gives rise to new sounds to which the auditory nerve is unaccustomed. In corroboration of this view, Reik has demonstrated that persons suffering with tinnitus have a lower blood pressure than that which is considered normal for healthy individuals.

Downey carried out a careful series of clinical investigations under Reik's direction, which he reported to the American Otological Society in 1912. In this report he was unable to confirm Reik's observation of a uniform lowered blood pressure in cases of tinnitus. In certain cases he succeeded in benefiting the tinnitus by the use of the suprarenal gland extract. On the whole, however, the impression we get from reading his article is that its use was a disappointment.

Finally, so far as those cases of very severe tinnitus are concerned, there can be no question that they have their origin within the brain.

Prognosis. To a large degree the prognosis of tinnitus aurium rests upon the proper location of the cause. At the same time it must be said that no positive prediction should be made, favorable or otherwise, in advance of treatment. Before instituting treatment a most careful inquiry into every possible condition within and without the ear is called for. Of all conditions within the ear, none is so common as a swelling and obstruction of the eustachian tube, and no cases offer greater encouragement for relief than those where the cause resides here.

Treatment. It is Politzer's belief that inflation by his method is superior to that by the catheter to relieve this condition There are many cases, however, where neither bag nor catheter is sufficient and where it will be necessary to make use of gradual dilatation by means of bougies. Great credit is due to one who was until recently an active worker among you, Dr. E. M. Holmes, for his perfecting and popularizing the nasopharyngoscope which bears his name. By this we have been able in recent years to recognize many pathologic conditions of the pharyngeal mouth of the tube. In our own hands direct treatment to the tube by means of this has far exceeded in value any other treatment which we have employed. Hand in hand with the treatment to the eustachian tube goes treatment to the nose and throat. Every one of you has had cases where relief of the tinnitus was promptly secured by removal of some obstruction or growth within the nasal chambers or in the nasopharynx, especially in the fossa of Rosenmüller. The same assiduous attention to the stomach and intestinal canal as well as to the tonsils and teeth is called for. We know of a case where the removal of a chronically diseased appendix cured the noise. In many cases the tinnitus gradually grows less as time goes on or the patient becomes more accustomed to it. This is particularly true in case of otosclerosis. Apart from the local treatment to the nose, throat and ear, much can often be accomplished by careful attention to diet, hygiene, etc. Alcohol undoubtedly increases the intensity of the noise. The same cannot be said of tobacco. Some years ago Reik carried out a series of experiments which showed that tobacco apparently had no effect upon the noise whatever. Internal medication is exceedingly variable in its results. Strychnin, in our hands, gives the best results. Iodid of potash we have used with benefit where the disease was located in the internal ear. Some most encouraging results have been obtained from the use of nitroglycerin. Bromide of potash and hydriodic acid we have repeatedly employed without satisfactory results. Reik, as a result of his experiments and deductions that there existed in all cases of increased intratympanic pressure a lower blood pressure, has employed the suprarenal gland with the view of raising the blood pressure. Used thus, the results were to him encouraging. This treatment has been curative or markedly beneficial in about three-quarters of all the cases, sometimes slow and only beginning after a period of delay. Possibly the effect of internal administration is cumulative.

Finally, so far as those severe and intractable forms of tinnitus are concerned which have their origin inside the cranium, no ordinary method of treatment is of the slightest avail.

In such cases careful consideration of more radical procedures is in order.

Destruction of the labyrinth does not offer great encouragement. Duel in 1915 reported to the American Laryngological, Rhinological and Otological Society a case where he had twice operated on the labyrinth without relief of the noise.

Even the still more radical, and what would seem far more encouraging operation of destruction of the auditory nerve, has proved a disappointment.

At the Ninth International Otological Congress, held in this city in 1912, Mr. Milligan, Dundas Grant and Mr. Lake were all opposed to this operation, stating that they had nothing but failures from it.

Dench has reported a case of persistent tinnitus where he destroyed the auditory nerve with temporary benefit, but when last heard from the noise had returned. As a much less radical measure, Reik at the otologic congress in 1912 reported a case where he ligated first the internal carotid artery and later the common carotid with complete relief to the noise, and also reported that upon Halsted's recommendation he has placed a permanent metal band upon the carotid artery with equally good result.

CONCLUSIONS.

Our study of the subject leads to the inevitable conclusion that many phases of it still remain to be cleared up; particularly is this true of etiology.

Of the several accepted theories to explain the origin of tinnitus, that of Reik's seems the most plausible. It is easy to understand how any factor can cause a dilatation of the blood vessels and so an increased flow of blood through the ear. This does not need to be within the middle ear itself. It can reside in the external auditory canal, as for instance, a foreign body or impacted cerumen pressing upon the drum. Yet even this theory, plausible as it is, hardly serves to explain the many well known variations of noises in the ear. Why, if they originate strictly in the middle or inner ear, should fatigue or overeating increase their intensity? Why, in two persons suffering from the same disease, should the noise be present in one and absent in the other? Or why, with no change in the objective findings, is the noise apt to become less intense as time goes on, if a vasomotor dilatation is present? Finally, the failures to relieve the noise by operations upon the labyrinth, or even by severing of the nerve itself, give us food for thought that its real source is not within the ear at all but in the brain itself.

So far as treatment is concerned, it is our feeling, as we have turned the subject over in our mind, that when we have failed to relieve it by attention to the nose and throat and the eustachian tube, together with such measures as may be necessary, addressed to the gastrointestinal canal, the outlook for therapy is not encouraging. We have spoken of success by means of drugs administered by the mouth; these cases undoubtedly are met with. We have taken occasion to inquire of Dr. Reik what his latest opinion is in regard to the use of extract of suprarenal gland. He did not hesitate to say that his early expectation had not been realized, although in a few cases he had gotten good results. Personally, we have had no experience with it. If the Reik theory is correct, it must follow that the use of a drug like this will have a beneficial influence, but operations, even such as loosening adhesions in the middle ear, cutting the tensor tympani, etc., and especially the larger and more extensive, such as cutting the auditory nerve, even if they give temporary benefit, are to be undertaken with great deliberation and only as a measure of last resort.

XIII.

POSTOPERATIVE TREATMENT OF THE RADICAL MASTOID OPERATION.

By J. A. Stucky, M. D.,

Lexington, Ky.

The topic as given to me by the Program Committee, to be considered in this symposium, would indicate that at least two other phases of this major surgical operation had been or were to be considered: 1. The indications for the radical mastoid operation; 2. The technic of the radical mastoid operation.

The postoperative treatment of the radical mastoid operation is of special interest to the surgeon because of the uncertainty of its duration, the annoying complications and results of neglect to patiently and perseveringly, almost daily for many days or weeks, treat the case, make it the operation to be most dreaded and avoided by the patient and otologist.

This is due to the fact that so many operators have had unsatisfactory results, even after long and careful treatment. As a basis for the after-treatment of the radical mastoid operation, it is assumed: 1. The operation was indicated; 2. A complete operation was done; 3. The tympanic orifice of the eustachian tube was closed, or so nearly so as to prevent retention of secretion and moisture in the middle ear cavity.

The two things that make any postoperative treatment uncertain as to time and results are: 1. An incomplete operation; 2. Neglect to so modify the bony and cartilaginous meatus as to produce the best facilities for drainage, ventilation and inspection, and failure to thoroughly remove the pathologic conditions in the different recesses of the middle ear cavity. The posttympanic space and eustachian tube are often a source of a purulent discharge and necessitate a protracted treatment.

Since my paper before this society, 1915, I have performed the complete radical operation on the mastoid in only a few cases, the modified operation as advocated then giving satisfactory results, except in cases where there was indication of extensive necrosis in the attic and mastoid process or intracranial involvement. Postoperative treatment begins as soon as the operation is completed, the external wound sutured, cavity dried and inspected.

The healing of the external wound is much quicker and more satisfactory if traumatism of the surrounding area is minimized. The unnecessary use of a number of hemostats and the too forcible and long continued use of retractors may so lower the vitality of the parts, as a result of bruising and stretching, that union or healing is seriously impaired.

I have used the various antisepticizing solutions in the operated area, during and after completing the operation, but none has given me as uniformly satisfactory results as a 1 to 1,000 solution of alphozone. This powder is readily soluble in water, is thoroughly antiseptic, nontoxic and does not interfere with the blood clot when this is used in the classical mastoid operation. This remedy is made of succinie acid and hydrogen peroxid and is said to be "as germicidal as mercury bichlorid, 75 times as germicidally powerful as carbolic acid, 100 times stronger than formaldehyde," etc., but the solution must be freshly prepared, as "when dissolved in or on contact with water alphozone combines with a portion of the water and undergoes a change known as hydrolysis."

Satisfaction or success in the postoperative treatment consists in rapidly obtaining and maintaining a dry cavity, covered by a vascular fibrinous dermatized membrane.

The completeness of the operation and refinement of technic has much to do with attaining this end, but notwithstanding the thoroughness of the operation and perfect surgical technic used, neglected after-treatment will result in disappointment or failure of the operation.

In some cases it is most difficult to keep down granulations, and granulations decrease hearing, especially if allowed to form on the internal wall.

The first dressing consisting of a folded strip of gauze, which had been saturated with 5 per cent iodoform emulsion and so applied through a large speculum as to touch every part of the cavity and tight enough to prevent any sagging of the fibrocartilaginous wall or contraction of the external auditory meatus; at the same time care must be used to pack evenly but not forcibly—the latter may result in interference with free circulation in the soft parts or displace the flaps made from the fibrocartilaginous canal.

The external dressing, consisting of pads of folded gauze wet in the 1 to 1,000 alphozone solution, snugly fitted around the external cartilaginous ear, over this absorbent cotton is applied and held in place by a bandage. This external dressing is allowed to remain for at least 48 hours, when it is removed and a similar but lighter dressing is applied. The packing in the cavity is not removed for four or six days after the operation, and this must be so carefully done as not to cause pain or bleeding or interference with the firm union of the fibrocartilaginous flaps.

The cavity is not irrigated but thoroughly dried with absorbent cotton, and again packed with 5 per cent iodoform gauze. In the second dressing I do not use the iodoform emulsion. At this time the sutures are removed and folded gauze placed over the posterior wound, held in place by the bandage. Best results have been obtained by rapidly reducing the amount of external dressing and doing away with the bandage—both are uncomfortable and retain too much heat and moisture. The wire screen mastoid protector devised by me some years ago, Fig. 1, I find most satisfactory and enables me to do away with the heavy dressing and bandage on the fourth or sixth day after the operation. After the second packing is removed from the cavity, which is usually on the sixth or seventh day, no further packing is used unless there is sagging or prolapsis of the metal flap, but instead the entire cavity is filled with powdered boracic acid and alphozone (six parts boracic acid to one part pulverized alphozone), two or three times a day, until the cavity is thoroughly dry and covered by a vascular fibrous membrane. For two or three days after beginning the use of the boracic acid and alphozone there is quite a free discharge of seropurulent or serous fluid, which fills the bowl of the external ear. This is removed with sterile cotton, the middle ear cavity dried with the same material by means of an applicator. The object being to keep the cavity as free as possible from heat and moisture, covered with the powdered boracic acid and alphozone, thoroughly ventilated and protected from infection.

Slow recovery, elevation of temperature or postoperative fever I have found to be more frequently due to absorption of proteids or toxins from the intestinal canal and not from sinus thrombosis. Most cases requiring the radical mastoid operation are more or less septic—the vitality and power of resistance is far from normal and to this condition we are to add the results of the anesthetic plus the operation.

It must be remembered that in septic conditions food not digested and assimilated thoroughly deranges the body chemistry, fermentation and putrefaction go on.

Ordinary bowel movement may remove some of this, but an empty alimentary canal does not mean a clean one; peristalsis is imperfect and the result of food putrefaction, and small particles of fecal matter remain in the folds and pockets of the canal, which give rise to toxins which easily find their way into the blood and lymph stream. For these reasons, immediately after the operation the patient is kept comfortable for at least 24 hours; usually the administration of one hypodermic of 1/12 or 1/8 grain of heroin will accomplish this. Free purgation, restricted diet (protein free), daily bath and gentle massage, fresh air, sunshine and rest.

The method I have outlined briefly is the treatment I have followed almost exclusively for the past eight years, because of the satisfaction given. The cavity is dry and dermatized usually in from three to six weeks.

Other methods, such as skin grafting, blood clot, bismuth paste and paraffin, etc., in my hands have not been satisfactory.

XIV.

TIC DOULOUREUX IN RELATION TO LATENT MAXILLITIS.*

By H. B. Lemere, M. D.,

Omaha, Neb.

Tic douloureux or trifacial neuralgia is characterized by sharp stabbing pains of intense severity, sometimes accompanied by facial spasms. The pain is usually referred to the distribution of the different terminal branches of the fifth nerve. An attack usually consists of several of these poignant, excruciating shooting pains, each short in duration and repeated at varying intervals. These stabbing pains may last for one-tenth of a second or more, or be in the form of an intense pain lasting as long as fifteen minutes, the average duration is less than a minute. The pain is entirely distinct in its characteristics from the pain resulting from a toothache or the ordinary supraorbital or infraorbital ache commonly called neuralgia. A pain that is continuous and throbbing in character cannot be classified as a tic douloureux. An acute sinusitis pain has no relation to trifacial neuralgia. The facial spasm is not a true motor tic which is purposeless, but is of the nature of muscular spasms elsewhere which accompany pain. It may be entirely absent, and replaced by the immobility of the face, or by the patient rubbing the face or by other forms of reaction to the pain. The attacks may occur at frequent intervals during the day or perhaps be several months apart. They rarely occur at night. Women and men are about equally liable, and either side of the face seems equally susceptible; over 90 per cent of cases occur after the age of 40, and 65 per cent of these between the ages of 50 and 70. Hugh T. Patrick gives the ratio of incidence as follows:[1]

The first or ophthalmic division is affected in 8.5 per cent.

The second or superior maxillary division is affected in 49 per cent.

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*From the Otological Department of University of Nebraska College of Medicine.

The third or inferior maxillary division is affected in 25 per cent.

Two or more divisions are affected in 17.5 per cent.

The clinical observations of the older writers attributed the neuralgia to anemia and to rheumatic, gouty and neuropathic predispositions or conditions. These general conditions we now generally find depending on a focal infection as the underlying cause. Patrick,[1] however, in his excellent article on the symptomatology of trifacial neuralgia, from which most of my statistics are quoted, states that, "In no case have I known treatment of a sinus or tooth infection or the extraction of carious or painful teeth to cure trifacial neuralgia," and he gives as reference Hajek's experience, that he had never seen tic douloureux in sinus disease. Patrick's article is based on the careful observation of 200 cases and, as no general or local condition is found as a causative factor, the deduction would seem to indicate an idiopathic etiology.

I recognize the weight that the opinion of such careful observers carries. Yet there have several years elapsed since the above statements were made, and the authors in considering the modern conception of the pathology of the nose and of the alveolar processes probably might revise their etiology if they were to write at the present time.

In the literature of the past ten years Beck[2] finds but three references to the pathology of trifacial neuralgia in which the writers have also been workers.*

Such profound pathologic changes as these represent, however, the terminal stage. The exacerbations of pain are probably caused by vasomotor reactions within the nerve sheath and ganglion and their effect increased by surrounding osteitis.

It seems to me that a neuralgia of this type is unlikely to originate of itself, and that it is worthy of careful study to determine whether trifacial neuralgia is idiopathic and primary, or whether it is secondary and due to local osteitis of the bone surrounding any of the terminal branches of the three divisions of the nerve. On this will depend the rationale of the treatment. If the neuralgia is primary, then attacks on the nerve are indicated; if it is secondary, the primary osteitis should receive first surgical attention in the hope that the nerve may return to normal, and that the general conditions resulting from the osteitis, viz., anemia, rheumatism, neurasthenia, etc., may also disappear. This latter will be a real cure with a return to normal. Any procedures which rely on a deadening or killing the nerve result at the best in only symptomatic relief of pain.

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*Postmortem examination of cases "that gave a history of long standing socalled severe face neuralgia show a definite shrinkage of the ganglion and its trunks. They appear paler than normal. Microscopic evidences are of a chronic neurogliar interstitial neuritis. The blood vessels are smaller. It appears as though the nerve was being starved of blood and is crying out for food instead of showing evidences of engorgement, which no doubt existed when the process first began and still does at certain periods of acute exacerbation."[2]

Adson[3] agrees with Patrick that nasal and dental surgery do not help this neuralgia. He says:[4] "The true case of trifacial neuralgia has never been relieved by sinus drainage nor by the removal of the teeth."

The accepted operative procedures among neurologic surgeons, according to Adson,[4] are as follows: Deep alcohol injections into the peripheral branches and ganglion, avulsion of the peripheral branches, or division of the posterior sensory root of the Gasserian ganglion. For this last operation he has devised an extremely delicate and exact operation. Pleth[5] reported several cases of keratitis resulting from injections of alcohol deeply into the Gasserian ganglion, and in one case there was loss of the eye. He recommends cervical sympathectomy.

Flint physiology, 1895, agrees with the modern surgical findings that if the nerve is divided posterior to the ganglion the nutritional nerves from the sympathetic to the cornea are intact. The cornea is, however, anesthetic and there is no muscular reaction of the lids to protect the eye from foreign bodies or trauma. The cornea is, therefore, liable to traumatic injury and infection which may result in blindness from opacity of the cornea or from loss of the eye.

A consideration of the following facts leads me to the conclusion that there is perhaps a very definite cause for these neuralgias.

The fifth nerve differs from other sensory nerves in the fact that its peripheral branches pass through bony canals in bones which are particularly liable to osteitis. The clinical findings show that each branch is attacked in proportion to the length of the branch so surrounded by bone, and also in proportion to the liability of the surrounding bone to inflammation.

The first or ophthalmic division of the fifth is the division least subject to attacks of tic douloureux. The frontal bone is not so frequently the seat of osteitis as the maxilla and the mandible, as the frontal sinus is much less liable than the antrum to chronic infections, and the frontal bone is removed from dental caries. The supraorbital branch runs through the supraorbital foramen, which has practically no length, and is often only a notch, so that it is surrounded only a ring of bone and has no canal.

The second division is the most frequently affected, and its infraorbital branch most subject to attacks of neuralgia. This branch passes through the infraorbital canal and groove and is surrounded by the bone forming the superior wall of the antrum for the entire length of that wall. The anterior terminal of the canal is the infraorbital foramen which emerges from the anterior wall of the antrum.

My findings in the examination of different specimens taken from both the anterior and the nasal walls of operated antra, show that generally there is a rarefying or a sclerosing osteitis accompanying a low grade or latent antrum infection. Added to this, the antrum is probably, of all the nasal sinuses, the one most frequently the seat of chronic infection. If to this factor is still further added dental pathology, the superior maxillary bone is seen to be particularly vulnerable to infections causing osteitis.

The inferior dental branch of the third division also runs through a long bony canal in the lower jaw, but as this bone is removed from nasal infections, and as the osteitis has to extend some distance from the alveolar processes to reach the canal, this branch escapes more often than does the infraorbital nerve.

Osteitis surrounding the different canals occurs therefore in about the same order of frequency as does the neuralgia, attacking the different branches. (See previously quoted table.)

The chronic osteitis and the neuralgia also both occur during the same period of life. They are least frequent in the first three decades of life, more frequent in the fourth and fifth, and most frequent in the fifth, sixth and seventh. Therefore, pathologically, anatomically and chronologically, trifacial neuralgia is closely associated with osteitis in the bones through which the terminal branches of the three divisions pass.

If these deductions are correct, the question resolves itself into treatment of the osteitis. Modern oral surgery is taking care of dental infections by partial or complete alveolectomy when neecssary, thus removing not only the offending tooth, but also the affected cancellous bone surrounding it. It remains for rhinologists to fill in the missing link by diagnosing and taking care of the osteitis resulting from either manifest or obscure antrum infections.

Tic douloureux is an extremely severe and persistent condition, and if attempts are made to relieve this condition by merely correcting nasal deformities these attempts will result in failure. Chronic infective processes come under an entirely different classification, and whether in the nose and mouth or elsewhere, should receive careful consideration. The surgeon, whether he be neurologic or rhinologic, must take them into account or his patient will inevitably suffer for his neglect.

It is useless to expect that removal or cautery of the turbinates or correction of nasal deflections will materially affect the condition of latent maxillitis except in so far as they indirectly improve the drainage of the antrum. This indirect effect, however, is not sufficiently reliable to be of much value in dealing with a chronic antrum involvement.

A latent maxillitis has certain unmistakable signs. They may all be present or only in part. They are:

1. Mucopus in the middle fossa. This mucopus is not necessarily yellow and creamy. It may be only scant and dry, forming a glazed scab covering the middle turbinate. Many latent streptococcus infections of the antrum give only a slight amount of clear, often glairy secretion. The maxillary opening and contour of the middle nasal fossa may be such that the infected secretion is not seen in this fossa but drains posteriorily into the nasopharynx.

2. The posterior pharyngeal wall usually gives evidence of the infected secretion, either by the lateral swellings posterior to the posterior tonsillar pillars, by follicular swellings covering the oropharynx, or by a dry glazed film covering the pharyngeal wall, or by masses of frank mucopus seen coming into view from above the uvula.

3. The middle turbinates are often granular on their anterior portion and there are sometimes polyps.

4. The density of the X-ray shadow is controlled by several factors which may be present.

(a) Osteoporosis or osteosclerosis.

(b) Osseous changes from concurrent alveolar diseases.

(c) Contents of the antrum. Thick mucopus producing a dense shadow, scant streptococcic secretion intensifying the shadow hardly at all.

(d) Thickness of the lining mucosa and presence of polyps.

When these underlying conditions are considered, the interpretation of the X-ray plate will give definite and reliable information. The shadows will have varying intensities, which I think can best be expressed by + signs, four plus being a practical opaque antrum and the other shadows grading down to a slight cloudiness, which is called one plus. If the cloudiness is still more indefinite, it may be recorded by an interrogation mark. Some antra with only faint X-ray showing have been the seat of latent streptococcus infection.

It is conceivable that an infection of the antrum with an essential atrophy of the mucous membrane and rarefaction of the bone might even appear clearer than normal. I have seen clinical evidence of this assumption, though I have never operated on such a case.

I attribute what success I have had in the following cases to through and through drainage, according to the technic I have previously described.[6] [7] [8] Unless it is understood that these latent or obscure maxillites so often do not necessarily consist of a frank empyema, there will be difficulty and failure in the diagnosis. It seems to me that the failure to recognize these low grade infections, and to provide relief, is responsible for the statements of neurologists and neurologic surgeons that nasal surgery is powerless to relieve tic douloureux.

As these cases of latent maxillitis are diagnosed and given through and through drainage in the treatment of tic douloureux of the second branch, I believe there will be less need of radical procedures on the Gasserian ganglion or its root.

Following are three case reports:

Case 1.—Aug. 25, 1919. Mrs. A. G., age 60. For past ten years has been subject to severe headaches and nervousness. Has been pale and had poor appetite. Two years ago she had sharp stabbing pain in the region of the left cheek. For past six weeks has had these attacks daily, each attack lasting about one minute. During this period patient has refused to eat, fearing to bring on an attack. She has lived on liquid nourishment. Examination: Teeth, none filled or crowned. No pyorrhea. Nose: Middle turbinate swollen. Scant catarrhal secretion. Pharynx: Scant dry film of mucopurulent secretion. X-ray: Teeth vital and sound. Right antrum clear; left antrum, + + +.

Conclusion: There is a trifacial neuralgia, increasing in severity, confined to the second division of the fifth nerve and most active over the infraorbital distribution. The primary cause is a low grade chronic infection of the maxillary antrum (latent maxillitis).

The left antrum was opened according to the author's technic[6] and through and through irrigation carried on for two weeks, when the patient returned home, eating normally and feeling well. At the time of operation ten minims of alcohol was injected into the infraorbital nerve. There was no accumulation of pus in the antrum. The anterior wall was thickened but very soft, and the mucous membrane of the antrum was thickened and pale and had adherent scant mucopus.

April 22, 1921. Twenty months after operation the patient's general health and appearance were excellent. She had had no return of her neuralgia except this March, when she had several attacks during one week. These attacks seemed to be coincident with an acute reinfection in her antrum. The opening beneath the inferior turbinate is now found to be free and a large sized eustachian catheter was used to wash out the antrum. The return was clear except for some flakes of mucopus. The next day the antrum was found to be entirely clear, and the attacks disappeared.

Case 2.—Mr. G. C. M., age 50, has had spasmodic, sharp, shooting, excruciating paroxysms of pain in region of left infraorbital branch of fifth nerve for past ten years. Attacks have been weeks apart, and last year had no attacks; five months ago had the flu, and since then has had attacks almost every week. In each attack the spasms came on every four to six minutes and lasted about an hour. The patient was very anemic and had been reduced to an extremely feeble state with marked nervousness. It was with difficulty that he could muster up courage to come up for examination, and he had to be supported on either side when he walked. He stated that he had sought relief and had been told of the operation for cutting the posterior nerve root, but had declined it on account of the possible after-results to the eye and to sensation.

Examination.—Nose: Both middle and inferior fossæ on both sides show slight mucopurulent secretion. No pure pus. Teeth: No alveolar trouble. Some pyorrhea. Pharynx: Glazed film of mucopurulent secretion. Tonsils: Lt., N.; Rt., +. Ears: Rt. canal filled with pus; Lt. memb. tymp. slightly reddened; no discharge. X-ray: Both antra cloudy. + + Sphenoids? Other sinuses clear. Rt. mastoid sclerosed; Lt., normal.

Both antra opened. Bony anterior walls very much softened. Bone and antral mucosa bled freely. No retention of pus in either antrum; mucous membrane in both showed softening and thickening resulting from pyogenic infection. Alcoholic injection of left infraorbital branch ten minims.

The writer's technic and after-treatment was followed with uneventful recovery. The patient returned home in about two weeks in good condition. Patient returned to Omaha in two months and reported doing a full day's work on the farm, which he had not been able to do for years. He had gained in weight and was robust and strong and his anemia had disappeared. He had a slight attack of neuralgia this spring, probably due to an acute reinfection of his antrum, but he reports his general health as being excellent with no further attacks.

Case 3.—Mr. D. F. C. December 8, 1920. Age 63. Referred by Dr. G. A. Young, with diagnosis of trifacial neuralgia of infraorbital branch, left side. Patient, a robust man, says he has been suffering from attacks of neuralgia with sharp excruciating spasms for the past two years. Have been so violent for the past three weeks that he has lost sleep and has an extremely drawn and worn expression.

Examination: Scant dry bloody scabs and mucopus in left middle nasal fossa, mucus in right middle nasal fossa. Tonsils: +. Teeth: Several stumps above and below. Pharynx: Seems clear. X-ray: Rt. antrum, +; other sinuses, +; Lt. antrum, +; other sinuses, +.

Dr. Young advised draining the antra. Operation: Ether, both antra opened. Bony walls on both sides seem to cut normally. On opening the antra the mucous membrane was found thickened and covered with adherent slimy, foul smelling, transparent secretion. The author's technic was followed at operation and for postoperative treatment. The dental stumps were extracted. The patient had a slight twinge of pain the first two days, but no paroxysms. He made an uneventful recovery and returned home in about two weeks, having had no further pain. He reports at writing of this paper as being in excellent health and has had no return of his neuralgia.

My conclusions are as follows:

Treatment of the maxillitis is the operation of choice because

1. It removes a dangerous focus of infection.
2. It preserves the function of one of the most important cranial nerves.
3. It safeguards the eye against ulceration of the cornea, as a cornea that is anesthetic is particularly liable to injury, even with the operation of the posterior sensory root, which leaves the sympathetic fibers of the ganglion intact.

In the treatment of tic douloureux we are dealing with a persistent condition, and therefore I present these cases merely as a preliminary report, as enough time has not elapsed to judge as to the permanency of results. I trust that in the future observations of others may be added as to the relationship of chronic maxillitis to tic douloureux.

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XV.

DICHLORAMIN TREATMENT OF MASTOID WOUNDS.*

By Ralph A. Fenton, A. B., M. D.,
Portland, Ore.

Otologists have long beaten about for methods of treatment of the mastoid wound which would abridge the duration of the healing process and the pain of dressings. Perhaps this summary of results in some recent cases may be of assistance to those who are not in permanence committed to older methods.

During 1916-17 the writer made intermittent use of chlorazene, in powder and in various weak solutions, with variable success. Dakin's solution was not available in proper form in hospitals at that time, and some dermatitis was noted, perhaps from too great chlorine concentration.

There was not much mastoid work in the advanced evacuation hospitals at the American front in 1918; but in the surgery of battle injuries of the face and head much experience was gained in the management of extensive lesions of cranial bones by chlorine disinfection. Methods of protection to uninvolved skin and minutiæ of administration of the Carrel-Dakin solution were quickly assimilated in the crowded dressing rooms behind the lines, in spite of failure of the Medical Department of the Army to give training in this technic before officers left the United States for France.

Three serious objections to Dakin treatment of head wounds were: Difficulty of retaining tubes in place; ineffectual flushing of tubes by busy nurses, and difficulty of avoiding maceration of the skin by aqueous chlorinated solutions. In war practice, however, these objections were far outweighed by the immediate cessation of bacterial activity and the very rapid healing. Late in the campaign, when primary suture after total excision of damaged tissue was at last being adopted from the French technic by many American surgeons, conservatism had to be maintained in head cases for cosmetic and functional reasons. But perfect results in selected cases reminded one of the perfect results in the "bloodclot" mastoids of Reik—an almost parallel surgical procedure.

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*Read before the Pacific Coast Oto-Ophthalmological Society, eighth annual meeting, Seattle, July, 1921.

Dichloramin-T in oily media was used in the A. E. F. wherever it could be had, especially in 1 to 4 per cent solutions in eucalyptol, for bony sinuses and fistulæ not susceptible of treatment by the aqueous solution. Eucalyptol was preferred by the orthopedists as a solvent because of its low specific gravity and high penetrating power in long channels. The eucalyptol solution was readily injected by means of tubes.

It was found that in applying oil-soluble dichloramin-T to the relatively shallow mastoid wound, however, the eucalyptol solutions often leaked out of the tubes or the suture line, or might even be smelt or tasted arriving in the throat via the eustachian route. Dichloramin treatment being different in principle from antiseptic treatment, this loss was deemed undesirable. Dichloramin makes human cells unpleasant food for microorganisms, and must therefore be present in a wound until the microbes stop multiplying. For this reason we have preferred to use chlorcosane as a solvent. This heavy, viscid hydrocarbon oil has been chlorinated so that it will freely take up dichloramin-T if slightly warmed. Its viscidity is such that it will not readily flow into small tubing.

The technic employed in these mastoids, which was modified from that followed in other lesions of cranial bones of traumatic origin, is as follows:

The bone cavity is mopped dry and oozing controlled. Two silkworm gut sutures close all but the lower fourth of the skin-periosteal wound. With a medicine dropper two per cent dichloramin-chlorcosane solution is now injected into the antrum and allowed to fill the wound. A few centimeters of narrow selvedged packing is soaked with the oil and placed in the skin opening, as a plug to prevent the wound contents from escaping, unless inflammatory pressure arises. One end of the oiled packing is spread along the incision. Another short oiled wick is carried through the external canal down to the membrana tympani. It should be clearly understood that these wicks should be very lightly inserted—never packed tightly—except of course where tight rolls are used to control sinus hemorrhage.

The wick in the ear comes out twelve to twenty-four hours after the operation and is not replaced. The canal should be kept dry by frequent swabbing with dry cotton. Dichloramin should not be used there any more because of the possibility of dermatitis in those cramped quarters.

The packing behind should come out in 48 to 72 hours (unless septic temperature indicates prior release of pressure by suture removal, moist applications, etc.) The wound will commonly show no more pus, but merely a bloody mucoid secretion, often somewhat scanty in comparison with that found at operation. The chlorine odor will replace the usual bloodclot or saprophytic stench.

Second and following dressings, made every day and later every third day, are done by very lightly swabbing out the wound with soft sterile cotton swabs, finishing with one or two swabs soaked in dichloramin-clorcosane. This is painless unless the skin edges are shoved apart. Half to one cubic centimeter of the oil should be dropped into the cavity as long as space exists, but oil should not be allowed to remain on the skin surface. This is the greatest cause of dissatisfaction with this agent; in sensitive skins erythema and dermatitis may be avoided by covering with a good zinc oxid ointment with mineral or lanolin base, and also by 50 per cent alcohol moist dressings.

Forty-five mastoids, in forty-three cases, have been tabulated in this study; all but two are "simple." (The detailed tabulation as exhibited at Seattle appears in the Transactions of the Pacific Coast Oto-Ophthalmological Society for 1921, including dates, initials and other necessary data from November, 1919, to June, 1921.)

Mastoids 1, 14, 15, 18, 27, 34, 35, 36 and 37 are excluded from totals regarding this technic because of general or local complications ranging from erysipelas and measles to pneumonia. The following averages are found in the remaining 36 cases:

Duration of postoperative temperature	2.8 days
Stitches removed after	4.0 days
Pus gone from wound after	4.8 days
Discharge gone from ear after	3.3 days
Left hospital after	6.4 days
Left hospital (33 cases excluding scarlet, erysipelas and cardiac cases) after	5.2 days
All moisture gone from wound after	16.0 days
Wound healed (smooth, painless, no scab) in	25.5 days

It will be noted that the length of time for pus discharge from the wound and for hospital and office dressings is very considerably reduced from that of the older methods with antiseptics and repeated packing. This technic of dressing is less difficult, and is practically painless to the patient, although it should be guarded against irritative phenomena. Plugging up a wound with oiled gauze or sewing it up tight around a tube may seem radical to those unfamiliar with the principles of chlorine treatment; but the results are so agreeable to patient and surgeon that it is hoped others will try the method out. Results will speak for themselves.

616 Journal Building.

Date	Initials	Age	Sex	Temperature	(days postop.) Stitches out	Pus gone from wound	Discharge gone from ear	Left hospital	Discharge gone from wound	Wound healed, dry, no scab
Case 1.—11/13/19.	P. P. J.	38	M	14	4	3	2	....	6	....
Case 2.—11/23/19	R. R.	14	F	6	4	4	3	20	24	32
Case 3.—12/30/19	R. H.	22	F	1	3	2	2	6	18	32
Case 4.— 1/ 6/20	I. L. G.	24	M	2	5	7	2	4	10	30
Case 5.— 1/ 8/20	L. W.	71	F	11	2	3	18	17	22	35
Case 6.— 1/15/20	E. K.	21	F	2	4	5	4	5	14	20
Case 7.— 2/25/30	B. J. K.	60	M	3	4	12	4	4	20	36
Case 8.— 3/ 1/20	B. D.	10/12	M	2	4	4	2	2	11	19
Case 9.— 4/ 9/20	C. A. J.	35	M	0	4	4	2	3	10	17
Case 10.— 6/18/20	G. V. W.	12	M	2	16	6	2	3	14	16
Case 11.— 6/21/20	T. B.	12	M	1	3	7	3	6	10	21
Case 12.— 7/ 7/20	E. W.	12	M	0	2	2	3	2	8	24
Case 13.— 8/ 4/20	P. C.	22	F	1	3	5	3	7	14	24
Case 14.— 8/16/20	E. B.	25	M	2	5	8	4	7	30	45
Case 15.— 9/16/20	H. N.	4	M	3	4	4	7	5	....	....
Case 16.—10/22/20	H. J.	7	F	3	2	4	6	7	18	20
Case 17.—11/20/20	M. L.	15	F	3	4	4	3	3	14	24
Case 18.—11/25/20	C. K.	2	M	3	2	4	4	3	90	90
Case 19.—12/ 4/20	K. J.	18	F	2	4	5	3	4	16	23
Case 20.—12/ 9/20	A. H.	18	F	2	4	3	2	6	20	31
Case 21.—12/20/20	G. S.	8	F	4	3	8	5	21	18	20
Case 22.—12/30/20	D. P. M.	23	F	7	2	3	2	10	9	10
Case 23.— 1/13/21	C. M.	6	F	2	4	4	2	5	11	29
Case 24.— 1/14/21	H. N.	8	F	2	3	7	6	4	24	38
Case 25.— 1/21/21	F. F. S.	46	M	1	6	4	6	6	19	22
Case 26.— 1/21/21	J. K.	2	M	0	4	3	1	4	16	20
Case 27.— 1/17/21	G. B.	rt. 3	M	28	3	31	26	10	43	44
Case 28.— 1/21/21	G. B.	left	....	28	2	5	4	4	16	26
Case 29.— 2/ 4/21	C. M.	7	F	7	4	5	2	4	22	32
Case 30.— 2/ 7/21	J. P.	7	F	3	4	4	2	6	20	34
Case 31.— 2/25/21	E. P.	6	M	2	3	5	3	4	24	29
Case 32.— 3/18/21	G. B.	7	M	2	4	4	2	6	29	34
Case 33.— 3/21/21	W. I.	7	M	4	2	6	3	5	15	20
Case 34.— 3/23/21	J. M.	6	M	2	4	7	4	5	....	....
Case 35.— 4/ 4/21	D. C.	7	M	4	2	4	3	7	40	43
Case 36.— 4/14/21	C. P.	rt. 7	M	14	4	3	2	....	9	10
Case 37.— 4/14/21	C. P.	left	....	....	4	4	3	....	10	11
Case 38.— 4/15/21	E. H.	2	M	7	2	11	6	13	25	27
Case 39.— 4/21/21	J. K.	76	F	0	5	3	2	7	18	20
Case 40.— 4/28/21	M. P.	7	F	2	4	5	3	5	20	22
Case 41.— 4/30/21	F. K.	23	F	3	4	8	3	10	22	28
Case 42.— 5/11/21	E. K.	7	F	2	5	7	2	6	15	20
Case 43.— 5/25/21	L. M.	7	F	1	5	6	3	4	23	21
Case 44.— 6/ 6/21	C. B.	8	F	1	4	4	1	3	10	13
Case 45.— 6/16/21	W. O.	7	M	7	6	2	1	10	11	12

COMPLICATIONS.

Case 1.—Fatal case of old C. C. O. M., converted into radical operation. In spite of skin sloughing, wound clean on postmortem. Death fourteenth day; leptomeningitis not of otitic origin, pneumonia.

Case 5.—Erysipelas of face and scalp on third day.

Case 8.—Mother tuberculous.

Case 14.—Zygomatic extension 20 days postoperative curetted, healed.

Case 15.—Left observation 17 days postop. Reported small fistula four months postoperative. Father tuberculous.

Case 18.—Sequestrum removed three months postoperative. Mother since died of tuberculosis.

Case 21.—Mastoid complicating scarlet fever.

Case 22.—Slight postoperative cellulitis of the neck.

Cases 27 and 28.—Zygomatic extension right side, healed by curettement 30 days postop. Recurrence where someone had done partial mastoid three years before.

Case 32.—Mastoid complicating measles.

Case 33.—Recurrence where someone had done partial mastoid four years before.

Case 34.—Postoperative treatment elsewhere; healing delayed 30 days by forgotten stitch.

Case 35.—Bronchopneumonia 20 days postoperative.

Cases 36 and 37.—Death 14 days postoperative from coexistent purulent meningitis. Mastoids clean on postmortem. Focal abscesses both tonsils.

Case 38.—Some sloughing of skin.

Case 39.—Mastoid ten days after acute bronchopneumonia.

Case 45.—Acute nephritis, mastoid and pneumonia due to tonsillar abscesses found at operation.

Mastoids 1, 14, 15, 18, 27, 34, 35, 36 and 37—nine in all—are excluded from totals regarding this technic, because of general or local complications as noted.

XVI.

ACUTE LACUNAR ADENOIDITIS.

By Samuel Salinger, M. D.,

Chicago.

Acute lacunar adenoiditis is pathologically similar to acute follicular tonsillitis with which it is frequently associated.

Because of this association and the fact that the tonsillar affection is more widely known and more readily diagnosed, the adenoiditis may frequently be overlooked entirely and run its course undiagnosed and untreated, with the possibility of recurrences and sequelæ.

Ordinarily, having diagnosed the tonsillitis, one is not likely to examine the nasopharynx. In fact, with tonsils and pillars acutely inflamed and swollen, it is not an easy matter to do a posterior rhinoscopy on account of the narrowing of the fauces and their heightened irritability. The presence of numerous yellow or white capped enlarged lymphatic follicles on the posterior wall of the oropharynx as well as along the nasopharyngeal fold should always excite suspicion of the process higher up and lead one to pursue the examination further.

Less frequently the adenoids may become acutely inflamed without involving the tonsils at all, or the process may occur in patients whose tonsils are out. These cases should present no difficulties in diagnosis, despite the apparently normal appearing fauces and oropharynx. All that is necessary is that one take the trouble to use the mirror or else examine the nasopharynx by anterior rhinoscopy, as will be described later.

Two cases in point will be briefly cited:

Miss K. T., aged 23 years, had her tonsils removed in June, 1917. For some time after, she complained of aching in the ears and received treatment, but with no permanent relief. A few days before appearing in our clinic she had a chill and a rise in temperature with severe pain in both ears and in the throat. The examination disclosed the following: Both membrana tympani were normal except for a slight retraction and a bit more translucency than normal. Nothing was found in the nose but a moderate swelling of the turbinates. After the application of adrenalin, the posterior nares could be made out and were seen to be full of a thick mucopurulent secretion.

The tonsils were absent, but the pillars were intact and not the least inflamed. The oropharynx was also normal. On inspection with the mirror the nasopharynx was found to be filled with a large mass of adenoids, which were markedly inflamed and covered with a thick, tenacious mucopurulent secretion. A partial cleansing revealed the depressions in the adenoid mass oozing the same type of secretion. Cultures later showed the predominant organism to be the streptococcus. The posterior cervical glands on both sides were enlarged and tender.

The temperature during the attack varied from 100 to 102.6 degrees F.

Under local cleansing treatment plus applications of argyrol, the condition cleared up and, despite our insistence, the patient elected not to have the mass removed, although it was easily the size of a hickory nut.

Case 2.—Miss N. R., aged 18 years, came in as a suspected acute mastoid. She gave a history of severe pain in the region of the tips of both mastoids radiating into the neck, elevation of temperature, general malaise and muscular soreness, and pain in moving the mandible. There was tenderness and enlargement of the posterior cervical glands on both sides. The external auditory canals and drum membranes were found to be absolutely normal. The tonsils were slightly enlarged and moderately reddened. The adenoids, on inspection with the mirror, were found to be large and lobulated and covered with an exudate which could be seen extending deeply into the recesses of the mass. This case also recovered within a few days.

The symptoms as described in these cases are substantially as found in various textbooks. There seems, however, to be some slight differences in the nomenclature of the disease.

Gottstein and Kaiser[1] describe it as acute follicular or lacunar adenoiditis and draw a distinction between it and simple catarrhal inflammation of the adenoid. They further mention a more severe type as "acute phlegmonous inflammation of the adenoid," in which there is an abscess formation that may rupture spontaneously or may burrow downward, presenting in the oropharynx as a retropharyngeal abscess.

Zarniko[2] considers three types of acute inflammation of the adenoids, the catarrhal, lacunar and follicular, although the distinction between the two latter is not made very clear. He mentions acute rhinitis, otitis media, epipharyngeal abscess and metastic infections as complications likely to ensue.

Kyle[3] describes it only as "acute nasopharyngitis," considering the inflammation as of all the tissues of the nasopharynx rather than the adenoids alone.

Grünwald[4] in his Atlas gives a very good picture of the disease, which he calls "acute lacunar inflammation of the pharyngeal tonsils."

Ballenger[5] presents a clear description of the disease and calls attention to reddening of the lateral pharyngeal folds. which are frequently studded with yellowish spots, indicating infection of the numerous lymphatic follicles in the oropharynx.

The etiologic factors as a rule are exposure to inclement weather, a general state of depressed vitality and infection with streptococcus.

Bryant[6] reports ten cases of streptococcic infection of the adenoids which were all cured by local treatment.

A consideration of the cases cited above brings forcibly to mind not alone the necessity of thoroughly examining the nasopharynx in every instance where earache is a symptom, but also the important fact that many adults possess large masses of adenoid tissue which have failed to undergo the regressive changes generally assumed to have taken place with puberty. It is surprising, if one will take the trouble to look, how many adults harbor appreciable masses of these vegetations. Practically no age is exempt.

Logan[7] reports that out of a series of 652 adenoidectomies 284, or about 40 per cent, were on patients between the ages of 25 and 59. He further cites another case in a patient of 64 years.

Cuvillier[8] saw two cases at the ages of 60 and 65 years. Solis Cohen[9] had one in a man of 70 years. Schaeffer[10] reports one at the age of 71 years, and Couetoux[11] another at 72 years.

Lewis[12] further quotes several more recent observers along the same lines.

It is important that this knowledge be given wide circulation, not so much on account of the acute attacks of adenoiditis (which are usually self limited and of no great severity), but because of their recurrence and the numerous more serious consequences which may follow in their wake. Current medical literature is full of references to the adenoids as the cause of nasal obstruction, middle ear affections, chronic laryngitis, and so far as laryngologists are concerned the subject is a closed book.

The general practitioner, however, I find, is somewhat lacking in knowledge of these facts, particularly with reference to adult patients. The principal reasons for this are the usual acceptance as dogmatic of the dictum that adenoids disappear with maturity, and the lack of skill in making examinations of the nasopharynx.

Physicians as a rule make their diagnosis of adenoids by palpating with the index finger in the nasopharynx, which, to my notion, is an unnecessarily painful and disagreeable procedure. It has been my custom, in teaching students, to demonstrate to them that when by reason of lack of practice on the part of the examiner or an unruly pharynx the posterior rhinoscopy cannot be successfully performed, the adenoids can clearly be made out by anterior rhinoscopy if the inferior turbinates have previously been well shrunk with adrenalin. Unless the turbinates are excessively hyperplastic or a very markedly thickened, deviated septum is present, the procedure can always be carried out. All that is necessary is that adrenalin be liberally applied the whole length of the turbinate and that the light be properly focused. By holding the patient's head bent a little forward of the erect position and directing the light along the floor of the nose or between the turbinal and the septum, and by coming a little closer to the patient than for the ordinary anterior rhinoscopy (bearing in mind that the focal distance of the mirror is a fixed point), the examiner is enabled to illuminate the posterior nares and roof of the nasopharynx. Of course, with the Kirstern light the problem of focusing is simplified. If the adenoids are very large and irregular, they can at once be made out by their very irregularity of contour, their pendulous position and the numerous lights and shadows produced by the examining light playing over the surface. If the mass be smooth, as frequently it is in adults, it can be outlined by having the patient say "k" or "cocoa," which brings the soft palate upward against the mass and alters its position. It will be noted at the same time that the reflected light moves with the mass. Where the adenoids are very large they may be seen to reach down to the level of the inferior border of the lower turbinal, and adenoids of any appreciable size nearly always stop the soft palate from reaching its proper height during contraction, as compared with normal.

This method, while difficult at first, can nevertheless be perfected with practice, and is particularly recommended for examining children, as well as adults.

A painful and disagreeable procedure, such as palpation of the nasopharynx, should be avoided because it leaves a horrible impression on the minds of the little ones which will alienate them from physicians in general and make future examinations difficult.

To conclude:

1. Adenoids in appreciable masses are of frequent occurrence in adults.
2. Acute lacunar adenoiditis should be thought of in cases of earache with fever where the ears are normal, regardless of whether the tonsils are absent or present, inflamed or not.
3. Diagnosis of adenoids should always be attempted by anterior rhinoscopy in preference to palpation in cases where posterior rhinoscopy cannot be carried out.

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25 E. Washington St.

XVII.

THE PROBLEM OF THE HARD OF HEARING AND ELECTRICAL HEARING DEVICES.*

By George E. Shambaugh, M. D.,

Chicago.

Otologists have been interested principally in the diagnosis and treatment of diseases affecting the organ of hearing. There is another problem of great importance that is the legitimate function of the otologist to help solve, and that is the problem of what can be done for those cases of increasing deafness which are not amenable to treatment. Otologists as a class have been slow to appreciate their obligation in these cases. The result is that those afflicted in this way have been left largely to their own resources in attempts to solve their problems. It is important that otologists take up more seriously the question of what can be done for these cases. The field is rather an extensive one, for it involves social and economic questions as well as the important one of what can be done to bridge over the chasm resulting from the defect in hearing. The busy otologist, with his interests centered on the diagnosis and treatment of diseases of the ear, finds himself handicapped by the lack of time necessary to carry the extra burden of solving these additional problems. Fortunately, there has grown up an institution, the comprehensive aims of which are to ameliorate the condition of those who are losing their hearing. This institution is the League for the Hard of Hearing, which aims not only to assist in bridging over the chasm created by the loss of hearing, but as well in solving the social and economic problems of these cases. Already there exists in several of our larger cities similar leagues to that which Dr. Phillips has been instrumental in organizing in New York. It becomes the duty of otologists to assist in every way possible in the organization of similar leagues throughout the country to take up the work at the point where the efforts of the practicing otologists cease.

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*Read before the joint meeting of the Chicago Medical and Chicago Laryngological and Otological Societies, Nov. 9, 1921.

Our work as otologists will continue to be chiefly in the diagnosis of ear disease and in the treatment of those cases where treatment is of assistance, but we should not neglect the important duty of giving proper advice to those cases where the condition does not call for local treatment. First of all, it is our duty to give advice regarding the value of taking up lip reading and to assist these patients in selecting proper devices which serve as aids to the hearing. It is in regard to the latter that I desire to make a few observations. A large number of such devices exist, some of which are of use in one type of deafness, some in others. Still others have no particular value, and some devices advertised as aids may even be a source of danger to the remnant of hearing. It is clearly the business of the otologist to be able to advise his cases intelligently regarding the use of these instruments, yet, as I have already pointed out, we have been negligent in not taking seriously enough this responsibility. We are all aware how hopeless it is for the patient unadvised to find out these facts for himself. Some of these devices, especially the "artificial ear drums," as well as certain electric hearing devices, are advertised to the public, making extravagant claims that are, to say the least, misleading. What are the facts regarding the value of these devices intended as aids to the hearing? I shall not attempt to discuss in detail all there is to learn about these instruments, but shall point out some of the outstanding facts about which as otologists we should be informed.

In the first place, it is hardly necessary to say anything much about the abused use of "artificial ear drums." Such devices are of especial assistance only in exceptional cases of chronic suppurative otitis media with destruction of the conducting mechanism in the tympanum, and then only, of course, where the defect in hearing is quite marked and involves both ears. The simple rubber disc with a string attached to assist in carrying it into the fundus of the canal, and by which it may be withdrawn, is all there is to this device. Usually the patient himself learns to adjust this so that it is lodged properly against the remnant of the stapes so as to improve the response of this important link of the sound conducting mechanism to sound impulses.

It is more especially in regard to the hearing devices intended for the nonsuppurative ear diseases to which I desire to call attention. There are in general two types of instruments intended for these cases. One type consists of a device intended to assist in collecting sound waves from the air and conducting them into the external meatus. These are the ear trumpets and speaking tubes. The other type operates on the principle of the telephone receiver with a pocket battery. These two types of instruments are not equally useful in all forms of deafness. In a general way, it may be stated that the ear trumpet and the speaking tube are of assistance in cases of obstructive middle ear deafness, as well as in those of primary nerve deafness. It is in the latter, however, where they find their greatest application. In the more advanced cases of nerve deafness the speaking tube is the only hearing device that affords any material assistance.

A great deal of confusion exists among otologists regarding the application of the electric hearing devices, of which there are a number on the market. A canvass of some of our leading otologists has brought out the fact that a few do not recommend these devices for any of their patients, while admitting that some of their patients get them and apparently find them of assistance, since they continue to use them. Most of the men consulted recognized no particular type of deafness where such devices are of special value. A few expressed the belief that there exists some difference in the type of case benefited by their use without, however, being very specific as regards just what type received more assistance. As a matter of fact, there are some very definite facts regarding the application of these electric hearing devices. The first outstanding fact is that they are of distinct help only in certain types of deafness and not in others. In this respect they differ from the application of ear trumpets and speaking tubes, which, as I have already stated, are of assistance in all types of deafness. In a conversation recently with a man who is himself hard of hearing and who makes and sells an electric hearing device, I asked whether he recognized any difference among the hard of hearing in the usefulness of his apparatus. His reply was so much to the point that I shall give in substance what he had to say. In the first place, he was very emphatic that a difference does exist. Some cases were very much benefited by using his device, while with others he found it of no special assistance. What was to me more interesting was that he was able to select the cases where it is was applicable before making any tests. For example, he had observed that a person who developed deafness after the age of 50 was not benefited unless the defect had its onset much earlier in life, and it had become very severe only later. In the second place, he asserted as a general principle that it was only in cases where the hearing was noticed to be better when the patient was riding on the cars that were benefited. We have in these statements what is apparent to all otologists, a rough differentiation of two types of deafness. On the one hand are the cases of pure nerve deafness, those where the trouble had its beginning after the age of 50, and on the other hand the cases of obstructive middle ear deafness, including especially the cases of bony fixation of the stapes.

In this connection it may not be amiss to point out the fact that a defect in hearing is always due to one of two conditions, either of which may be acting separately in particular cases, or both may be active in the same case. In the first place, there is the defect in hearing caused by some obstruction to the sound impulses reaching the nerve of hearing. The simplest example of this type of trouble is where the canal of the ear is occluded by a plug of cerumen. Anything which causes fixation of the sound conducting mechanism in the middle ear produces a defect in hearing by hindering the transmission of sound impulses from the outer air to the end organ of hearing in the cochlea. This includes the several types of middle ear disease as well as the condition known as primary fixation of the stapes. It is the latter process which is the principal cause of the hearing defect in most cases of hereditary deafness.

The second cause for defective hearing is where the nerve of hearing fails to respond normally to the impulse of sound waves. This is called nerve deafness. This condition may develop primarily, but it frequently develops secondary to long standing obstructive middle ear deafness.

The otologist finds no difficulty in differentiating these two types of deafness, especially where they develop independently of each other. Confusion is sometimes caused by the mixed types of deafness—that is, where there is an interference with the sound impulses reaching the nerve, as well as a defect in the nerve itself. More difficulty is experienced in distinguishing the several types of obstruction in the sound conducting mechanism.

I have rarely seen a case of pure nerve deafness which received any considerable assistance from the use of an electric hearing device. Cases of deafness coming on after the age of 50 are with very few exceptions cases of primary nerve deafness. Of course, not all cases of primary nerve deafness have their origin late in life. A few of these patients begin to lose their hearing in middle life at the time when bony fixation of the stapes usually begins to appear, or even in childhood. The diagnosis of such cases presents no particular difficulty to the otologist.

It is in the cases of obstructive middle ear deafness where the electric hearing devices are of benefit, and it is particularly in those cases where the obstruction is due to primary fixation of the stapes that they are especially useful, because it is in these cases as a rule where the defect becomes so severe as to render the use of a hearing device of practical value. Increased bone conduction seems to be essential, and in most of these cases the symptom of paracusis Willisi (the ability to hear better in a noisy place) is present. It does not matter that in these cases of long standing obstructive deafness degeneration in the cochlea sooner or later develops, as is particularly conspicuous in otosclerosis. The associated nerve deafness is no contraindication to the application of the electrical device, because there continues to be an increase in the bone conduction resulting from the fixation of the stapes, so long as there is any function left in the cochlea. It is easy to understand why even in these advanced cases of otosclerosis such devices continue to be of assistance. The cochlear defect begins by destroying the upper part of the tone scale, which is of little use in the perception of hearing for the voice. The lower part of the scale is preserved until the very end, and this is the part of the scale used in conversation. The increased bone conduction for the lower part of the tone scale continues, and therefore the usefulness of the electric hearing device continues, even in the presence of an extensive nerve degeneration. I can see nothing but harmful results possible from the use of a noise making apparatus which is often urged upon the patient as a means of developing his hearing powers.

XVIII.

THE BACTERIOLOGY AND HISTOPATHOLOGY OF THE ADENOIDS.*

FROM THE DEPARTMENT OF PATHOLOGY AND BACTERIOLOGY OF
THE UNIVERSITY OF ILLINOIS, COLLEGE
OF MEDICINE, CHICAGO.

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Adenoids have been discussed for many years with reference to their hypertrophy and resulting obstructive symptoms and to their rôle as a portal of entry in tuberculosis. One phase entirely neglected has been the bacteriology. The following data represent the first systematic bacteriologic studies of the selected parts of the nasopharynx, the vegetations or adenoids. While many investigations of the bacterial flora of the nasopharynx with special reference to the carrier problem have been made, such work has always been carried out with cultures obtained with a swab which can at best touch the surface of the vegetations. These studies cannot be accurate in determining the extent the adenoids may harbor bacteria for like in the tonsils the flora of the crypts is different from that of the surface. To obtain more accurate knowledge of the incidence and numbers of the more common microorganisms and to ascertain the extent these organisms flourish in the recesses of the nasopharynx, a study was undertaken of the extirpated adenoids. Histologic studies were made to bring out the cellular changes, if any, to the presence of the bacteria and to correlate any particular pathologic condition with the character of the bacterial flora.

The material was obtained from children in the laryngologic ward of the Cook County Hospital during the months from April to November, 1920. The patients, ranging from five to fifteen years of age, were afebrile, with no acute inflammatory condition of the respiratory tract, and presented tonsils and adenoids with varying degrees of hypertrophy. The adenoids were removed under general anesthesia, in most instances with the La Force adenotome.

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*Read before the Chicago Laryngological and Otological Society, October 3, 1921.

The vegetations when well removed consisted of a soft mass of lymphoid tissue, varying in size from ½ cm. to 1½ cm. in all diameters. Practically all presented folds of tissue leaving one to five or more clefts of varying depth. In many specimens the folds had become partially united, leaving pits or slits from a few millimeters to a centimeter in length and depth. These pits resembled the tonsillar crypts, but as a rule were not as deep and did not tend to be narrower at the orifice than at the base. Most of the adenoids presented both folds and such cryptlike depressions. Many revealed only the folds, while a few were entirely pitted with the crypts. Gross evidences of pus were not encountered, but occasionally the crypts were filled with soft yellow fatty debris which microscopically consisted of amorphous material, cholesterol crystals and a few epithelial cells, leucocytes anad bacteria. Direct smears from the surface and depths of the vegetations revealed varying numbers of cocci and bacilli and cellular debris. The cocci appeared in short chains, often in pairs, spherical like staphylococci and streptococci, frequently lancet shaped like pneumococci. The bacilli were of different types, some like diphtheroids and others small pleomorphic, like influenza bacilli. Spirilla were observed in a few instances. The cells were chiefly desquamated epithelium, both of the squamous and cylindrical type, and leucocytes of the small and large mononuclear type, and a few of the polymorphonuclear variety.

The adenoids were collected in sterile gauze and cultures made from them in one to four hours. Macerated specimens were discarded. With a wire loop the surface of the vegetations was scraped and inoculated upon media, and then another culture was made with material obtained with the loop from the depths between the folds and the cryptlike depressions. The adenoids of 103 children were thus cultivated upon unheated blood agar and heated blood agar for the influenza bacillus. The adenoids of 100 other children were similarly studied upon Loeffler's serum medium for the diphtheria bacillus.

Every adenoid gave positive growth. The cultures from each adenoid showed several organisms, regardless of the size or structure of the vegetation, occasionally one organism predominating, rarely, however, in pure culture. The incidence of the various bacteria found are indicated in Table 1.

Hemolytic streptococci (1) occurred in 61 per cent of the cultures of the depths of the adenoids and in 58 per cent of the cultures from the surface. In 18 instances these organisms were quite numerous, and in three were found in practically pure culture. It is noteworthy that in the specimens with crypts these streptococci were usually more numerous than in the vegetations without such structures. In their behavior toward carbohydrates and in their pathogenicity for rabbits and mice they correspond with few exceptions to the streptococcus pyogenes group. Streptococci of the viridans type (2) were encountered in 89 per cent. On the basis of carbohydrate fermentation 30 per cent of the strains were of the streptococcus mitis group, 64 per cent of the streptococcus salivarious, 6 per cent of the streptococcus fecalis. Streptococcus mucosus was noted in 3 per cent. Large moist green colonies corresponding to the socalled Mathers coccus of the recent influenza epidemic occurred in 17 per cent. Pneumococci were found in 65 per cent, in four instances in practically pure culture. Classified according to agglutination with specific type 1, 2 and 3 serums, 85 per cent of the strains were 4, 13 per cent type, 3, 2 per cent type 2. Influenza bacilli (3) were identified and isolated from the chocolate medium in 40.9 per cent, frequently in large numbers. They were hemoglobinophilic, pleomorphic and showed the distinctive property of symbiosis. Diphtheria bacilla (4) were recovered in 12 per cent from Loeffler's medium, often in large numbers, and in two instances in pure culture on this medium. Two strains were toxin producing, while the remainder were relatively avirulent. Diphtheroids occurred in 30 per cent and were differentiated by their morphology and inability to ferment the common sugars. The staphylococci appeared in 60 per cent, chiefly as the albus type. Gram negative cocci, consisting chiefly of the micrococcus catarrhalis, micrococus pharyngis siccus and a few like meningococcus, were present altogether in 79 per cent. The Friedlander bacillus was found in 14 per cent and the B. fusiformis in 20 per cent of the ten anerobic cultures.

The flora of the nasopharynx, as revealed by cultures obtained with the swab, was compared to the flora of the surface and depths of the adenoids. The hemolytic streptococcus was found in 40 per cent of 25 persons in the cultures from the swabs, while the excised adenoids yielded larger numbers in 60 per cent of the same individuals. The greatest incidence and numbers of bacteria occurred in the depths between the folds and in the crypts of the vegetations, lesser numbers on the surface epithelium and least upon the swabs of the adenoids in situ.

The excised faucial tonsils of the same individuals were similarly compared and found to be somewhat different in their flora. Hemolytic streptococci occurred in 95 per cent of 21 pairs of tonsils, often in large numbers, as compared with fewer numbers in 71 per cent of the adenoids. Influenza bacilli were found in 53.9 per cent of 115 pairs of tonsils, whereas they were encountered in 40.9 per cent in the vegetations. Both adenoids and tonsils yielded the same percentage of diphtheria bacillus (12 per cent), but in considerably larger numbers in the latter. The diphtheroids, on the other hand, were present in only 17 per cent of tonsils, while the adenoids gave positive results in 30 per cent. From these results it can be stated that the tonsils harbor greater numbers of hemolytic streptococci, influenza bacilli and diphtheria bacilli, while the adenoids favored the lodgment of the Gram negative cocci and the diphtheroids.

The flora of the nasopharynx was further investigated in children whose adenoids and tonsils had been removed and compared with a similar group in which the adenoids and tonsils were present. Hemolytic streptococci were found in 11 of 27 individuals without tonsils (40.8 per cent), as compared with 27 of 48 with tonsils (56.3 per cent); influenza bacilli in 8 of 29 tonsillectomized (26.5 per cent), and in 17 of 43 nontonsillectomized (39.3 per cent). Pneumococci were found in about the same percentage (32.5 per cent) in both groups. The numbers of all of these organisms were decidedly fewer in the tonsillectomized children. It would seem therefore that the removal of tonsils and adenoids diminishes the incidence of dangerous bacteria, but by no means causes their disappearance. The remaining pitted follicles of the oro- and nasopharynx undoubtedly continue to harbor these organisms.

Histologic sections were made of the adenoids from 75 individuals, all of which had been studied bacteriologically with a view of determining if any distinctive cellular reaction resulted as a response to the presence of any particular bacterial flora. The first striking feature is the hyperplasia of the lymphoid tissue, with no other very remarkable changes. The degree of hyperplasia varied in different adenoids. The condition appeared as large follicles with active germinal centers showing many mitotic figures. The small lymphocyte was very predominant, with an occasional endothelial and a few polymorphonuclear leucocytes in their midst. In a few instances the endothelial cells seemed to have proliferated, forming small islands of large cells. The follicles made up the greatest part of the vegetation, except in the smaller adenoids, where the diffuse lymphoid tissue equaled or exceeded the area covered by the follicles.

The folds and cryptlike depressions were lined with epithelium under which lie the lymphoid follicles. The epithelium was chiefly of the stratified columnar ciliated epithelium variety, merging occasionally into the stratified squamous near the ends of the vegetations. The columnar epithelium frequently showed a tendency to become modified, particularly in the deeper portions of the clefts and cypts, into two or three, or even a single layer of columnar cells, especially in specimens with very large follicles located immediately underneath. At times it was difficult to see any epithelium; the lymphoid tissue apparently lies exposed with no cellular evidences, however, of any ulcerative process. Throughout just underneath the epithelium polymorphonuclear leucocytes and plasma cells could be seen in varying numbers. Between the folds and in the depths of the crypts debris consisting of disintegrated epithelial cells with an occasional polymorphonuclear leucocyte and lymphocyte, was often encountered. A few specimens revealed hyaline, pink staining material containing a few cells and bacteria, which material when examined in the fresh specimen was made up of amorphous fatty masses and cholesterol crystals. The epithelium about such masses was usually thin and often squamous in character. In sections stains by the Gram-Weigert methods bacteria were surprisingly few. An occasional diplococcus or bacillus was found on careful search on the epithelial surface or free in the cellular debris, but was not detected in the depths of the lymphoid tissue.

Connective tissue was variable in amount, appearing in larger amount in the vegetation showing considerable diffuse lymphoid tissue. Plasma cells and eosinophiles were often seen in the connective tissue, especially about the blood vessels. At the base of the vegetations mucous glands frequently occurred.

No striking pathologic changes could be ascribed to any flora or organism. The adenoids with deeper folds and many crypts, particularly those containing amorphous and cellular debris, as a rule harbored larger numbers of bacteria, especially hemolytic streptococci. For the most part histologically the specimens were uniform in their appearance, regardless of the character of the flora. Every adenoid presented evidences of chronic inflammatory change no greater in degree than the changes that are present in the lymphoid tissues in close proximity to the mucous membranes of the normal individual. In none were found acute cellular reactions to the presence of the organisms that lie in close contact with the vegetations.

In the great majority of individuals bacteria apparently lie dormant as saprophytes in the adenoids without exciting acute local or general symptoms. These organisms may stimulate gradually hyperplasia with plasma cell and slight leucocytic infiltration. Like in the tonsil, a variety of dangerous bacteria exist without harm to the individual as long as the resistance of the local tissues and of the body is good. In the course of acute infections involving the respiratory passages, as in the exanthemata, influenza and other diseases, the local resistance of the tissues becomes lowered in some unknown way by the primary virus or parasitis bacterium, and the general resistance is diminished by the acute toxemia. Under these conditions the saprophytic organisms no longer lie dormant but become invaders growing rapidly in neighboring tissues, into the blood and lymph stream, with the resultant serious complications. In this way the streptococcus, influenza bacillus and other bacteria give rise to the secondary infections of the lungs, heart, the middle ear, the accessory sinuses, meninges and other parts of the body.

From these studies it is well to recognize that the nasopharyngeal vegetations should be regarded not only as hyperplastic obstructive masses but also as foci like the tonsils, where dangerous bacteria are lodged. This conception is to be borne in mind in connection with the treatment of diphtheria carriers where the removal of the adenoids as well as the tonsils frequently terminates the carrier state. In the treatment of the metastatic infections, such as endocarditis and arthritis, the adenoids should be regarded as possible foci of infection. Although probably not as important in this respect as the faucial tonsils, the adenoids are to be looked upon as a portal of entry of many acute and chronic diseases.

THE INCIDENCE OF COMMON PATHOGENIC BACTERIA IN THE ADENOIDS.

Organism	Number of Individuals	Per cent Positive
Streptococcus hemolyticus	103	61
Streptococcus viridans	103	89
Pneumococcus	103	65
Type 2	103	2
Type 3	103	10.3
Type 4	103	52.7
Mathers coccus	103	17
Staphylococcus	103	60
Gram negative cocci	103	79
B. influenzæ (Pfeiffer)	115	40.9
B. diphtheriæ	100	12
Diphtheroids	100	30
B. mucosus capsulatus	103	14
B. fusiformis	10	20

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SOCIETY PROCEEDINGS.

THE CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Annual Meeting, May 2, 1921.

THE PRESIDENT, DR. ALFRED LEWY, IN THE CHAIR.

Symposium: The Future of Otolaryngology in Chicago."

DR. ALFRED LEWY said that with the idea in mind that this was a forward looking society, one of the members of the council, who was a good friend of his and of the society, suggested this program in lieu of the customary retrospective presidential address usually given at the annual meeting. Among our members are quite a number of men of wide reputation as investigators, teachers and clinicians, several of whom had consented to lead the discussion, each taking up briefly some particular phase of the subject. The first to speak would be Dr. J. Gordon Wilson, his subject being "The Training of Students Toward Greater Accuracy."

DR. J. GORDON WILSON believed the future of laryngology in Chicago depended on the ideals of those practicing the specialty, on the standards of proficiency they endeavored to maintain. This society, composed of the leading specialists in otolaryngology, ought to play an important part in determining this future. Various views would no doubt be presented, for the pathways of progress are many. There was, for example, a recommendation of the council in regard to research in otolaryngology. Such required ample time and necessitated some sacrifice. It required laboratory facilities and presumed the acquirement of preliminary knowledge and technic. Few have the opportunity or training to explore unknown fields, but one field necessary to the advancement of this specialty was open to all—namely, careful observation of cases and accurate records. Everyone acknowledged the importance of accurate observation and, in reports, the need of careful statements of facts in simple language, but such are too often wanting. Accurate observations of conditions was not easily acquired. Symptoms as given by the patient were often inaccurately noted. Those who have frequently to look up case reports know the inadequacy of many of these. This was a profitable field in which each could assist. This society should insist, so far as it can, that papers presented and afterwards published be devoid of errors in observation and statement.

Dr. Wilson pointed out the fact that they were sadly deficient in postmortem reports in otology. Surely in this society they ought to be able to show some work on the microscopic examination of ear cases carefully tested during life.

Dr. George E. Shambaugh stated that there are two phases of work in connection with graduate instruction in otolaryngology which have usually been confused. One is the work which a man should do preparatory to taking up the specialty. The other has to do with providing short review courses for those already established in the specialty. These are often called "brush up" courses. These men do not care to spend a long period of time on such work, but wish to have in a short time an opportunity to add to their knowledge regarding some particular subject. The phase of work of preparing for special practice is quite a different proposition. These men need prolonged, careful study, lasting at least a year. Very little effort has been made to provide this sort of training in this country. The men entering our special field have been forced to rely upon the short, intensive, "brush up" courses which are suitable especially as review courses for those already established in the specialty. This is the same sort of work that they have found when they went abroad. Very few found the opportunity of doing the fundamental work by filling the place of clinical assistant for a long period in some properly equipped and properly organized outpatient department. The sort of preparation that these men have received has been, to say the least, very unsatisfactory. Proper preparation cannot be obtained by listening to lectures and clinics and by filling a notebook full of medical facts. When the medical student has finished his undergraduate course he should be through, in a large measure, with "spoon fed" instruction. His development from that time on depends largely upon his own initiative, and the most substantial assistance we can give him is to provide proper facilities and opportunities for doing the right kind of work, but it should be up to him to work out his own salvation under such conditions.

Dr. Shamhaugh called attention to the fact often overlooked by our men seeking preparation in the special field, that the foreigners have never relied upon the taking of courses. These courses were provided to satisfy the demands of the American students, who somehow have been imbued with the idea that by paying out money to an instructor for ladeling out facts for his consumption, he will get that which he is too indolent to work out for himself. The American Medical Association in its Council on Medical Education has taken up seriously this question of providing a proper minimum standard of preparation for those desiring to enter the special fields of practice. The Committee on Otolaryngology insists on the principle that the preparation for special practice should be put on the basis of genuine graduate instruction, in which facilities are provided for doing the work, but the student must work in a large measure independently under supervision but not by course taking. The plan approved of consists of one and one-half years' full time work. For the first year this work must be done at one place. One-half of each day to be devoted to the clinical study of cases in a properly equipped and properly organized outpatient department, such as exists in connection with many of our Class A medical schools. In this work the student is required to learn how to examine cases and how to make proper diagnoses. It is only in the latter period of the course that instruction in operating is provided. The other half day is to be spent in the laboratory of the university working on the fundamental sciences, especially the anatomy of the ear and nasal cavities. Here the same type of work is insisted upon: The student must work out the subject himself under supervision but not by course taking. After this year of fundamental training it is suggested that as far as the opportunities permit these men shall secure positions as interns in special hospitals or as residents in otolaryngology in general hospitals where they shall spend the minimum of one year. Those who are unable to secure such opportunities should spend the last six months necessary to fill out the minimum eighteen months' requirement by continuing the work of the first year, or by taking such courses in various medical centers as would meet the approval of the institution where they have taken their first year's work, or by working as assistants in the office of some established specialist. On the completion of this work it is suggested that the institution where the first year's work has been taken should issue a certificate stating that the student has had proper preparation for undertaking special practice.

As for providing facilities for this work, these already exist in connection with many of our Class A medical schools. It has been generally accepted that 100 men turned out each year would amply fill all the legitimate requirements to take care of all the ear, nose and throat work in this country. For some years the country has been swamped with general practitioners, many of them rather unsuccessful, who have come to clinics and learned something about two or three operations of the nose and throat, and on the strength of this have gone back as specialists. Dr. Shambaugh thought one could easily say that if all of these men had proper training in the examination and diagnosis of cases so that they could appreciate the proper indications for surgical interference, only a small percentage of the operations which they are now attempting to do would be recommended.

The Facilities for Postgraduate Work in Chicago.

Dr. Otto J. Stein said this was a difficult subject for him to speak upon because they had as yet not formulated any definite plan upon which to carry out their own work. Many critics have attacked postgraduate teaching with the idea that such schools tend to create specialists, but this he wished to emphatically deny. He believed this idea had arisen from the fact that many men attend the postgraduate schools and then return home and practice some one specialty, but he believed that this did not mitigate against the idea that these schools make specialists. They do not give men certificates saying that they are specialists, but give them a statement of attendance saying that they were in the school for a certain length of time taking courses in that particular line.

Medicine has changed from the homogeneous science of older days to a more complex science, split up in heterogeneous groups, and otolaryngology has become a separate science, but this has not yet been recognized by the medical schools, and they still insist on teaching undergraduates otolaryngology. This often gives students an idea that they know something about this specialty, they attend a postgraduate institution after gradauting, and if they like this specialty they study it and go back and practice it. Dr. Stein thought that medical science is now large enough for a student to devote all his time to the fundamental branches without going into any of the special branches, and was inclined to criticise any schools that give undergraduates instruction in otolaryngology. The surgeon could, of course, speak of the complications of the nose and throat as he sees them and could touch upon the anatomy, and the man in general medicine could touch upon the complications as he sees them, but no special instruction in any specialty should be given to undergraduates. He believed that steps were now being taken to have this matter remedied.

Dr. Stein believed the otolaryngologist himself is much to blame for the attitude of the general public in reference to this specialty. The specialty is too much split up, one man not doing any work on the larynx, another not doing bronchoscopy, another not doing mastoid operations. He believed that if a man wanted to be a specialist he should be thoroughly trained and able to understand, treat, discuss and teach all phases and branches of the specialty.

Another point touched upon by Dr. Stein was that the general practitioner should not be blamed for working in this specialty, for he has been given the impression in his undergraduate days that he is receiving some training in this line. He often is better qualified to do the work than the men who are associated with one in the same hospital and community, who are recognized as great surgeons, or orthopedists, or pediatricians, but who dabble in this specialty without ever taking a special course in the work.

Dr. Stein believed the future of this work rests entirely on two phases of the subject: First, the work must be thoroughly standardized, as the outline given by Dr. Shambaugh showed to be possible. Second, each one in the specialty must be thoroughly equipped and able to take out tonsils, or remove a turbinate, or do mastoid operations or anything else that may be required, and not limit himself to any one or two phases. Much depends upon the institutions by which we will create proper material from which these men will profit.

The Future of Defects of Speech in Chicago.

Dr. Elmer L. Kenyon stated that disorders of speech are as inevitable as disorders of the nose, or of the ear, or other anatomics—physiologic disorders of the body—and their treatment just as essential. The field is just as definitely a medical field as is the field of otolaryngology in general. One trouble is that this field of effort does not fit in with the conventional tendencies of specialization. Some disorders of speech are capable of being fully understood only by the otolaryngologist. Either the neuropsychologist should unite with the otolaryngologist and handle the subject jointly or else there should be special training in neuropsychology and otolaryngology for the especial purpose of handling this subject. During the reecnt war the Government placed responsibility for treatment of disorders of speech chiefly on the department of otolaryngology, and that helped to place responsibility for such disorders on this specialty.

A few years ago the Section on Otolaryngology of the American Medical Association created a permanent committee on the deaf child, and a year ago the section extended the work of that committee to include not only the deaf child, but the deaf adult, and also disorders of speech. It seemed to Dr. Kenyon that this was likely to prove to be an important step forward.

As to the present status of the work in the United States among physicians, so far as the speaker knew it, in Philadelphia, where Dr. Makuen worked so long and so well, no physician has taken up his work. Mrs. Steel goes on with Dr. Makuen's clinic in the Philadelphia Policlinic and takes care of disorders of speech so far as possible in the same way as when he was living. In New York, Dr. Scripture has gone to Europe and left Mrs. Scripture in charge of the speech work in the Vanderbilt Clinic. A few years ago, Dr. J. Sonnett Greene decided to go into the work of disorders of speech as an exclusive specialty. He started what is known as "The New York Clinic for Defects of Speech," and is occupying an old residence for the work. This clinic has had much publicity, has interested many of the influential people in New

York and has a large clinical patronage. It has no medical school connection. In Boston, for some years, Dr. Walter B. Swift has been doing speech work. His public work now consists chiefly in going to different parts of the country at different times, where he establishes for a period of about six weeks a center of instruction for lay teachers in the treatment of defects of speech. This is one of the means by which teachers in our schools in various parts of the country are now being instructed to a certain degree to take care of disorders of speech. In St. Louis, Dr. Max Goldstein has included disorders of speech in the work carried on by the Central Institute for the Deaf. At the University of Wisconsin, in Madison, Dr. Smiley Blanton is giving excellent instruction in this subject. In Chicago a clinic in defects of speech has been conducted by the speaker at Rush Medical College for about eighteen years. Instruction has long been given to the undergraduate medical students and is now beginning to be offered to graduate students of otolaryngology.

Dr. Kenyon thought that what was especially needed in Chicago was laryngologists who were willing to practice in this field. From the educational standpoint what was required were facilities or giving a certain minimum of instruction for the undergraduates in all medical schools, a certain larger minimum for the prospective specialists in otolaryngology and neurology, means for instruction to lay workers, and especially means for instruction to physicians who intend to specialize in disorders of speech. It seemed to him quite unlikely that all the medical schools in Chicago could ever have a clinic in disorders of speech. Instructors are certainly at present wholly inadequate, and clinical patients are likely to be diverted to the educational speech work in the public schools.

The way to handle the situaiton in Chicago, he believed, was to found an adequate institution which shall handle this field in a large way, from the treatment, research and the educational standpoints. This institution should furnish undergraduate instruction for all the medical schools in the city, for the graduate schools, for specialists in defects of speech and for lay workers. Such an ideal institution, Dr. Kenyon said, should be directed by the combined educational medical interests in Chicago. Only when such institutions have been established here and elsewhere, and the question of knowledge and treatment have thus become standardized, will this field of practice begin to take its rightful place in service to mankind.

The committee of the Section on Otolaryngology of the American Medical Association hopes in another year to bring forward a program which will encourage the development of this field.

Dr. Norval H. Pierce thought the definite plan of postgraduate instruction outlined by Dr. Shambaugh was practical and ingenious, as utilizing the possibilities of medicine as it is known in this country, but how is that plan to be carried out? It seemed to him that the whole subject of postgraduate instruction in Chicago is tinctured by our national beliefs. Democracy is of necessity diffuse in its efforts. It lacks definite scope. Dr. Smith may start a postgraduate school, Dr. Brown may start a hospital, and Dr. Green may even start an undergraduate school, all with different purposes and ideals. What we need is coordination and this, he thought, should take place through the university. He did not believe that postgraduate instruction would advance perceptibly if left in the hands of separate interests, but felt that coordination is sure to come. The Illinois Charitable Eye and Ear Infirmary is an example of our democratic incoordination. Dr. Pierce believed the men in that institution are, for the most part, doing the best they can and that they are guided by sane and practical policies, but thought it would never amount to anything as a teaching and research institution as long as it was a little comet circulating in an orbit of its own. It should be under the jurisdiction of the university. The university has authority, it has the administrative ability, and is a cogent body that can and will direct its parts on known idealistic lines. He expressed his great disappointment and sorrow that they had never been able to place the Illinois Charitable Eye and Ear Infirmary under a university administration, because it is used largely for the purposes of personal advancement in operative technic. The assistants come there not in order to help along and forward the general purposes of the institution. They come there to gain experience, and as soon as they are instructed, or as soon as they get as much as they think they can out of the institution, they either leave or they become disgruntled because they are not made chiefs of service. A course of instruction is now being started for the interns, but without system. In a haphazard way one doctor speaks on something today and somebody else speaks tomorrow—perhaps on the same subject. There is no coordination, no real directing of the students, which should be done by heads of departments or by some controlling body. This university plan would prevent the entrance to the teaching staff of incompetent men, and he considered this a very important function of the university control. Such a plan would stabilize the institution; courses would be outlined and given by heads of departments who would be responsible.

Dr. Pierce believed the only possible way to carry out the plan outlined in Chicago is to put the various eye, ear, nose and throat institutions under university control.

Dr. Pierce did not agree, and did not believe that any teacher would agree, with Dr. Stein in his suggestion that we eliminate instruction of every kind concerning the eye, ear, nose and throat from the undergraduate course. It would be impossible to do so because of the hospital examinations for interns. They ought to know something of these diseases and of the examination of these organs when they go into practice.

As to what the society was going to do to advance its professional interests, in the past the "old wheel horses" have been the ones who carried on the programs, but it is now up to the young men to "carry on," and carry on better than the old ones did. He thought it a deplorable fact that the programs of the medical societies are not much better now than they were twenty years ago. There is a lack of scientific research, too much superficial clinical work, too little individual thought and too little attention to the borderline matters of our specialty. We should all be as much interested in the science of our specialty as in the things that gain us our daily bread. Otherwise we are bound to degenerate. Physiology ought to be just as important as the pathology or the treatment of various kinds of diseases, and if this spirit can be implanted or activated we are sure to advance. If not, if the society is to meet just to hear about the removal of turbinates, catheterizations of the eustachian tube, or operations in general, the specialty is going to degenerate and will not come up to the standard which Dr. Pierce believed will be set in the very near future.

Dr. J. Holinger said he had been interested in the subject of advancing otology for thirty years. After thorough discussion, three fundamental requirements were put down. First, it is necessary to have a large clinic. The most important cases are rare. Only a large clinic can furnish an adequite number to be worth analyzing. The second requirement is a large and well financed laboratory with sufficient instruments and sufficient space for men to work in. The third important requirement is ample fresh postmortem material so that pathologic material can be secured in the early hours after death has occurred. If the profession is unable to fit out and support such a clinic, the general public should be called upon to help. The public should be educated to the importance of such work. The splendid work done in the Vienna clinic was only possible through the combination of a large clinic, good laboratory facilities and ample fresh material for postmortem examination. In the United States neither Siebenman nor Wittmaack would have had the opportunity of doing the important work that they have given to the profession. In going before the public and interesting them in the fact that these things are necessary to the further advancement of science, we will also interest the public in the things we are trying to do in their interest. They will recognize the difference between really scientific effort and quackery.

Dr. Joseph C. Beck said that in the past twenty years it had been his custom to take men and women into his clinic and train them for a year or more in the work of the clinic. During this time he has trained twenty-three men and women in this way. He realized that this work had some defects and intended to give it up in October, because he felt that what Dr. Shambaugh and the other members of the committee of the American Medical Association had recommended was the best that could be had in this country or anywhere else. In studying abroad he had watched the clinics over there, and if the plan proposed and outlined by Dr. Shambaugh goes through, we will have their method "beat off the earth." The method that is employed in Europe, especially for Americans, does not compare with this new plan. We have the universities, the laboratory facilities, the large outdoor departments, and all we need is to have the men connected with the university give their time to the work. Plenty of postmortem material can be secured if we can get rid of the crooked undertakers who prevent us from obtaining postmortems. The truth is that the profession is too busy to do the kind of work that Siebenman and many others over there do. There are plenty of men here who have the ability, but they are too busy with other things, not always practice, to do such work.

If the method outlined by Dr. Shambaugh is put into effect, Dr. Beck felt sure that a man can become a very well trained specialist in this country. Dr. Dean of Iowa City has a method of taking only five men at a time, and in that manner of training they can develop better than in any of the European clinics. In Vienna or Berlin they do not train the Americans as they do their own men but train their assistants a great deal as we now propose to do. By taking out the second or third assistant for a night or two of pleasure it is sometimes possible for the American student abroad to get an opportunity to do an operation, but seldom otherwise.

Dr. Beck agreed with Dr. Wilson that the taking of accurate histories is very important and that very few specialists do this. It is also important to follow up the cases as much as possible and make additional notes from time to time.

Another thing in touching upon the future of otolaryngology was that one should keep in better touch with the other branches of medicine. There is not enough reading done of articles dealing with borderline subjects. Dr. Beck called attention to what the Institute of Medicine of New York is doing in an attempt to interest the specialists in allied subjects, by furnishing comprehensive abstracts, such as has never been done before. He urged that the members of the society support this movement so that it would be carried on.

In his clinic at the North Chicago Hospital, in the future Dr. Beck and his associates will do only the "brush up" teaching for the men already trained as otolaryngologists, as outlined by the scheme of the committee of the American Medical Association and particularly by Dr. Shambaugh. Almost all of the men and women that Dr. Beck has trained in the past have been taken in charge by otolaryngologic friends of his, where they have served for a year or more as assistants and associates before going into practice for themselves.

Dr. Beck thought the facilities for postgraduate work in Chicago were excellent and that it would not be at all impossible to have an abundance of fresh postmortem material from that source also.

Dr. George W. Boot said he was not so much interested in the education of the specialists as in that of the undergraduate. He was sure it was a mistake to say that the undergraduate should not be taught anything about the eye, ear, nose and throat, for a man could not be taught too much about anything. It is a mistake to think that if these things are taught, a man will not refer work to his teacher, for the reverse is the case. The more he knows, the more cases he will recognize as needing the care of a specialist. Rush Medical College requires for graduation 1.2 major hours; Northwestern, 16 hours clinical and 96 hours dispensary work; Loyola, 0.8 of a major for ear, nose and throat work; P. & S. requires 60 hours' clinical and 18 hours5 dispensary work. Dr. Boot believed Northwestern had the best plan of all. If he had his way he would dispense entirely with the didactic work and most of the clinical work. Anything that can be given in didactic work can be learned just as well from a textbook, but he would like to have the students do real work in examining noses and throats, not their own, but those of the dispensary patients.

Dr. Boot agreed with Dr. Pierce that it was a crime the way the Illinois Charitable Eye and Ear Infirmary was managed, but believed this was largely due to the men who managed the place and the heads of the departments. He also thought the way the Cook County Hospital is managed is another crime. There is a constant stream of outdoor clinical material going there—enough material to supply all the classes that go through, but the only class that takes advantage of it is that from Loyola. He tried to get the classes from the College of Physicians and Surgeons to go there but did not succeed.

He believed Dr. Holinger's idea of a large institution where there could be many patients and abundant apparatus was a good one if it could be put into practice, but the obvious place for this is at the Eye and Ear Infirmary.

As to the necessity for accuracy of diagnosis brought out by Dr. Wilson, he heartily agreed. He had very unsatisfactory experiences along this line in hunting up histories of cases indexed as brain abscess at the County Hospital a year ago. Many of the histories were dictated by very capable men but were very incomplete, in many instances no statement as to location of the abscess being given.

Dr. Boot hoped that a different type of undergraduate teaching would be available in Chicago in the near future, and felt sure that if this could be brought about the future of oto-laryngology would be much brighter.

Dr. Shambaugh (closing) expressed himself as much pleased with the discussion. The subject had many angles which cannot be touched upon in a short discussion. A great mistake was being made in many places by allowing interns in general medicine to acquire the technic of nose and throat operations. These men had no opportunity of learning how to recognize the proper indications for such operations, and when they go out into practice one heard on all sides of the indiscriminate slaughter of the tonsils as well as of the turbinal bodies and operations upon the septum.

Dr. Shambaugh stated that in the outpatient department at Rush Medical College, he has for several years been carrying out a plan similar to that which the report of the American Medical Association outlined, for providing training in oto-laryngology. He has had a motto framed and hung in the outpatient department which reads as follows:

> "Three essentials for the successful practice of oto-laryngology, in order of their importance:
>
> 1. Proper respect for the patient and interest in his welfare.
>
> 2. Ability to make diagnoses and to recognize the proper indications for surgical interference.
>
> 3. Skill in operative technique."

He insists that this relationship must always be kept in mind in providing instruction for those preparing to take up this work.

THE CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday Evening, October 3, 1921.

THE PRESIDENT, DR. ROBERT SONNENSCHEIN, PRESIDING.

DR. I. PILOT (by invitation) addressed the society on

"Histopathology and Bacteriology of the Adenoid."

(ABSTRACT.)

The adenoids have been discussed for many years with reference to their hypertrophy and resulting obstructive symptoms and to their rôle as a portal of entry in tuberculosis. One phase entirely neglected has been the bacteriology. While many investigations of the bacterial flora of the nasopharynx with special reference to the carrier problem have been made, such work was carried out with cultures obtained with a swab which can at best touch the surface of the vegetations. To obtain more accurate knowledge of the incidence and numbers of the more common organisms and to ascertain the extent to which these organisms flourish in the recesses of the nasopharynx, a study was undertaken by Dr. S. J. Pearlman and Dr. Pilot of the extirpated adenoids. Histologic studies were made to bring out the cellular changes, if any, to the presence of the bacteria and to correlate any particular pathologic condition with the character of the bacterial flora.

The excised adenoids obtained from children 5 to 16 years of age consisted of lymphoid tissue made up of several folds and frequently revealed cryptlike depressions like the tonsils. Cultures were made upon blood agar and Loeffler's serum from the surface and from the depths of the clefts and crypts.

Every adenoid gave positive growth. The cultures from each adenoid revealed several organisms, regardless of the size or structure of the vegetation, occasionally one organism predominating; rarely, however, in pure culture. The incidence of the various bacteria found is indicated in the following table:

Organism	Number of individuals	Per cent positive
Streptococcus hemolyticus	103	61
Streptococcus viridans	103	89
Pneumococcus	103	65
Type 2	103	2
Type 3	103	10.3
Type 4	103	52.7
Mathers coccus	103	17
Staphylococcus	103	60
Gram negative cocci	103	79
B. influenzæ (Pfeiffer)	115	40.9
B. diphtheriæ	100	12
Diphtheroids	100	30
B. mucosus capsulatus	103	14
B. fusiformis	10	20

The flora of the nasopharynx as revealed by cultures with a swab was compared to the flora of the surface and depths of the adenoids. The hemolytic streptococcus was found in 40 per cent of 25 persons in the cultures from the swabs, while the excised adenoids yielded larger numbers in 60 per cent of the same individuals. The greatest incidence and numbers of bacteria occurred in the depths between the folds and in the crypts of the vegetations, lesser numbers on the surface epithelium and least upon the swabs of the adenoids in situ.

The excised faucial tonsils of the same individuals were similarly compared and found to be somewhat different in their flora. Hemolytic streptococci occurred in 95 per cent, B. influenza in 53.9 per cent, B. diphtheria in 12 per cent, and diphtheroids in 17 per cent The flora of the nasopharynx was further investigated in children whose adenoids and tonsils were removed and compared to a similar group in which the adenoids and tonsils were present. In the tonsillectomized, hemolytic streptococci occurred in 40 per cent, B. influenza in 26.5 per cent, pneumococci in 32.5 per cent. In the nontonsillectomized, hemolytic streptococci were found in 56.3 per cent, B. influenza in 39.3 per cent, and pneumococci in 32.5 per cent. The number of all of these bacteria was decidedly fewer in the tonsillectomized children.

Histologic sections were made from the adenoids which had been studied bacteriologically. No striking pathologic changes could be ascribed to any flora or organism. The adenoids with deeper folds and many crypts, particularly those containing amorphous and cellular debris, as a rule, harbored large numbers of bacteria, especially hemolytic streptococci. For the most part histologically the specimens were uniform in their appearance, regardless of the character of the flora. Every adenoid presented evidences of chronic inflammatory changes no greater in degree than the changes that are constantly present in the lymphoid tissues in close proximity to the mucous membranes of the normal individual. In none were found acute cellular reactions to the presence of the organisms that lie in close contact with the vegetations.

From these studies it is well to recognize that the nasopharyngeal vegetations should be regarded not only as hyperplastic obstructive masses but also as foci like the tonsils where dangerous bacteria are lodged. This conception is to be borne in mind in connection with the treatment of diphtheria carriers where the removal of the adenoids as well as the tonsils frequently terminates the carrier state. In the treatment of the metastatic infections, such as endocarditis and arthritis, the adenoids should be regarded as possible foci of infection. Although probably not as important in this respect as the faucial tonsils, the adenoids are to be looked upon as a portal of entry of many acute and chronic diseases.

Dr. Samuel Salinger read a paper entitled

***"Acute Lacunar Adenoiditis."**

*See page 220.

DISCUSSION.

Dr. D. J. Davis said a few words regarding the ideas and principles underlying the data presented.

Two parts of the gastrointestinal canal are of interest in connection with infections. Passing downward from the mouth to the rectum, if one makes cultures at frequent intervals, one will find two regions with maximum numbers of bacteria. There will be relatively a small number of bacteria at the lips and, passing down, they will increase very greatly, so that there will be found a maximum in the nasopharynx. In the region of the esophagus they decrease rapidly, and in the stomach there are relatively few bacteria. On passing down into the gastrointestinal canal, the numbers gradually rise, another maximum being attained in the region of the ileocecal valve and upper colon. From here the number of bacteria again diminish toward the rectum. This is also true as regards the incidence of pathogenic organisms and infections. Infections are very common in the nasopharynx region, then they are relatively uncommon until the lower part of the small bowel, and especially the region of the appendix, is reached, where again many infections appear.

We may parallel this incidence of bacteria and infections if we consider the amount of lymphatic tissue along the gastrointestinal canal, the maximum appearing in the pharyngeal region, then diminishing in the esophagus and stomach and again gradually increasing in the intestine in the region of the ileocecal valve. As to the meaning of this, Dr. Davis thought the body had developed this lymphatic mechanism in order to protect itself against the bacteria which live in these localities. The lymphatic mechanism presumably is a defensive one. Wherever it is found in the body it appears to be of use for protective purposes. The distribution of lymphatic tissue, for example, in the hilum of the lung, in the mesentery, in the groin, etc., would lead one to suggest that it is protective, and presumably that is the purpose of this tissue as it appears in tonsils and adenoids and in the gastrointestinal canal.

On the other hand, while the body has developed this lymphatic mechanism to protect itself, the bacteria are equally adaptive and have altered themselves in such a way as to attack the very mechanism the body has developed to protect itself. Streptococci, apparently by preference, frequently attack lymphatic tissues in the throat. The typhoid bacillus in the bowel evidently does the same.

Dr. Davis called attention to a peculiarity of the lymphatic tissue in the submucosa of the gastrointestinal canal. It has efferent channels but no afferent channels. It is located just beneath the mucosa, where it can readily absorb material and there is no need of afferent channels. There seems to be no question about the function of these lymphatics. Bacteria can practically always be found in the centers of these glands in the normal state. A few bacteria no doubt occasionally get through into the deeper lymphatics and thence into the circulation, where they usually die, but at times may cause infection.

The speaker considered the carrier question of importance in connection with the data presented by Dr. Pilot. The adenoids are of great importance in this regard, and one should always think of this location as a place where certain dangerous bacteria may reside for long periods of time.

As this study has progressed, they have come to realize that many parts of the body have developed their own flora. And as they investigate this point more in detail, they find that even very limited localities have their own individual bacteria. The bacteriology of the surface of the tonsils and of the crypts is very different, and the bacteriology of the adenoid may be quite different from that of the tonsil, or the pharynx, or the larynx.

Dr. Davis felt that much work is still to be done. Studies of this sort have just begun. For example, little is known of the anaerobic flora of the respiratory tract, and especially of the tonsils. He cited the fact also in this connection that it was not known until very recently that the influenza bacillus is one of the very common organisms of the respiratory passages, present often in individuals normally as well as in those suffering with various kinds of infections. It was largely because of the lack of knowledge of its distribution that it was so long thought to be the cause of influenza epidemics.

Because of the fact that new methods are being devised constantly, it is necessary to revise bacteriologic data frequently. This necessitates more or less constant reinvestigation. However, this work of Dr. Pilot's on the adenoid appears to be the first systematic study of the bacteriology of this organ ever made.

Dr. George W. Boot was impressed with the large percentage of patients who had tonsils and adenoids removed and still gave cultures of the same organism as when the tonsils and adenoids were present. This doubtless explains why in so many cases when the tonsils and adenoids are removed there still remains an enlargement of the lymphoid islands in the pharynx, which sometimes becomes very annoying.

Dr. Salinger called attention to the fact that his patient had enlargement of the postcervical glands. Dr. Boot thought this rather peculiar, as his idea of the lymphatic glands was that the adenoids drained into the same set of glands as the tonsil. He asked if anyone else had noticed the drainage into the postcervical glands.

Dr. John Edwin Rhodes said that this field of investigation was not available when he was a student, and he was much gratified to hear such a paper as that of Dr. Pilot. He considered the society was very fortunate in having such a paper presented, showing the painstaking work of the essayist in connection with the study. He felt that the profession should avail themselves of all such things in an effort to trace down the bacteriology of disease, and with the laboratory facilities of this time that was possible for every practitioner. He had been much edified and benefited by the papers.

Dr. J. Holinger wondered why if the diphtheria bacilli were in the crypts and folds of the adenoids they did not make trouble more frequently. The statement had been made that the children whose adenoids had been examined had no fever, no sign of acute inflammation and no local or general indications of the presence of these bacilli. No streptococcus hemolyticus or any of the virulent organisms had made any symptoms. He asked if there was any possibility of explaining these facts, or did the profession have to go back to the old expression of "genius epidemicus," which had meant much many years ago but which they thought they were rid of.

Dr. Harry W. Woodruff, Joliet, said he would not attempt to discuss the subject from the standpoint of bacteriology but was interested in the point made in the examination of adenoids in children. He had always felt that using the finger in the examination for adenoids was almost as severe and difficult as removal of the adenoids, and he had used the method suggested by Dr. Salinger for several years.

Dr. Alfred Lewy thought that very few realized the amount of intelligent industry necessary to get together the mass of data presented by Dr. Pilot, and expressed great interest in Dr. Davis' analysis of the work, especially in his observation that bacterial flora are most abundant where lymphatic tissue is, and the fact that infection occurs most frequently in those localities. He asked Dr. Davis (realizing that it was a philosophic rather than a practical question) whether it would not be as well to say that the infection was more frequently manifested in those points, and whether or not recent investigations had thrown any light on the cause of such apparent localization. He thought that as specialists we were likely to center our attention too closely on local conditions and not examine sufficiently into the general condition and environment of the patient for causes of nonresistance to infection.

Regarding Dr. Salinger's paper, referring particularly to acute infections of the nasopharynx, he thought it was rarely necessary to subject the patient during such acute conditions to attempts at posterior rhinoscopy, as he believed nearly every case of tonsillitis was associated with nasopharyngitis, either location being primarily inflamed. Ordinary sore throats are commonly nasopharyngitis, as indicated by the inflammation along the salpingopharyngeal folds. In his opinion chronic nasopharyngitis is the most common inflammation found in the upper respiratory tract.

MAJOR SHERMAN, M. C., U. S. A. (Camp Sam Houston, Texas), felt that the society was doing wonderful work and expressed his personal thanks for being allowed to attend the meetings. During the last year Major Sherman had been impressed with the large number of adenoids found in men in the army. There are now more boys of eighteen and nineteen than before the war, and the number of adenoid cases is large. The question of examining these cases naturally needs consideration as to why the patients have the same infection after the adenoids are removed. He had found some form of nasopharyngoscope which allowed a full view of the adenoid mass and had found no difficulty in using this, even in very young children. By this method it was not difficult to find exactly where the masses were. In his opinion, the reason there was recurrence was because all of the mass was not removed. It was interesting to see how many times when an adenoid was removed, a small walled off abscess was found within the mass.

DR. A. A. HAYDEN emphasized what Major Sherman said concerning the use of the nasopharyngoscope in looking for adenoid masses, either after or before the operation. This could be done almost without pain, and an excellent view of the entire region could always be obtained. He felt that everyone used the finger very frequently, because it was a convenient thing to do, but this was open to the very valid objection of alienating the affections of one's patients.

Dr. Thomas Faith said that when Dr. Salinger spoke of disseminating this knowledge to the practitioners in general, he felt one point might be added, namely, that these acute adenoid attacks explain many of the febrile attacks very young children often have. While this is recognized by laryngologists it is very frequently not recognized by practitioners in general. He believed that many of the socalled attacks of gastrointestinal disturbance in children could be explained as attacks of adenoiditis.

Dr. Faith believed that in disseminating the knowledge of the presence of adenoids in adults, the knowledge ot the acute attacks occurring in children might accompany it to advantage.

Dr. Harry Pollock called attention to the fact that some years ago one of the members read a paper on tuberculosis of the adenoids, and he was surprised that Dr. Pilot had not mentioned finding any tuberculosis in the 115 cases examined. This condition was quite common, especially in the presence of enlarged glands.

In regard to Dr. Holinger's question as to why the children with these large, diphtheria infected adenoids showed no symptoms—in examining school children Dr. Pollock had found as high as 15 to 25 per cent of the cultures sent to the Health Department showed diphtheria carriers, but the children were perfectly healthy. At that time they were not doing the Schick test, and he asked the Doctor what relation the diphtheria carriers now had to the Schick test. Recently there had been quite an epidemic of Vincent's angina. Dr. Pilot mentioned finding the fusiform bacillus, but said nothing about the spirilliform, and Dr. Pollock was surprised that he had not mentioned finding this in the tonsils and adenoids.

The speaker felt that the best way to locate large adenoids in adults was by means of the nasopharyngoscope, but children who had their tonsils and adenoids removed will not hold still and permit this, so it is usually necessary to make a digital examination. In adults in a few cases where adenoids were suspected, where the tonsils and adenoids had been removed but patients complained of discharge of mucus, one must think of Thorwaldt's disease, which could be mistaken for adenoiditis. They had recently had two cases in which they found Thorwaldt's disease, and after opening the bursa the patients got well. One patient, the daughter of a prominent surgeon, had large cervical glands. She had the tonsils enucleated, but the glands kept on growing and were diagnosed as tubercular glands. There was no trouble in breathing, but on examining with the nasopharyngoscope they found large masses of adenoid tissue. These were removed and the glands gradually disappeared. Their experience was that the posterior cervical glands would usually enlarge from the adenoids. In his opinion the adenoids drain through the posterior cervical glands and the tonsils through the anterior.

Dr. Pollock wished to know whether the children in whom the diphtheria bacillus was found gave a positive reaction to the Schick test.

Dr. E. P. Norcross said that he saw many children with acute conditions where the site of the trouble was in the postnasal space. In many of these cases the tonsils were not inflamed, the only evidence of a throat infection being a red pharynx and postnasal discharge. He believes that the adenoid is very often the site of acute inflammation in children.

Dr. I. Pilot (closing the discussion on his paper) said that the incidence of the organisms in tonsillectomized patients was frequent, but one should remember that there was considerable adenoid tissue still present in the nasopharynyx, all of which was pitted, and these follicles continued to harbor various bacteria in children who have had their tonsils and adenoids removed.

Regarding the question of diphtheria carriers, the number of positive cutlures obtained was 12 per cent of 100 children, and it was indicated that of the 12 strains only two proved to be virulent. In an extensive study made at Johns Hopkins Hospital by Guthrie, Gelien and Moss similar results were obtained, in which positive results were present in anywhere from 10 to 18 per cent of children. There, too, the strains were tested for virulence, and only about 11 per cent were found to be virulent, so the actual number of diphtheria carriers could be narrowed down to those carrying virulent diphtheria bacilli. Their data seemed to indicate that the carriers of avirulent bacilli were harmless and should not be treated as dangerous unless the cultures were tested for virulence.

Organisms of the streptococcus hemolyticus group, B. influenzæ and others that were quite virulent could exist in the nasopharynx without causing any symptoms on the part of the person, depending upon the resistance of the individual and also upon the invasive properties of the organisms. The organism might be virulent, but its power of invading the body was another property, which must be considered in determining the actual danger of the organism. The more invasive type of streptococcus hemolyticus due to infected milk from sore throat are organisms which are a little different in their reactions and have chiefly this invasive property. An individual infected by this particular strain is dangerous to another individual, while an individual carrying the ordinary streptococcus hemolyticus, which does not have this particular invasive property, is not dangerous.

Dr. Pilot had made no attempt to present any complete histopathology or bacteriology. The demonstration of evidence of either active or latent tuberculosis with strains of tubercle bacillus in the tissues constitutes a big problem in itself. Each of the organisms was studied and considered as a separate problem and no reference to the tubercle bacillus and other organisms was made because the bacteriologic study is not yet complete. The fusiform bacilli encountered were those found in anerobic cultures of ten specimens. The spirillum was encountered in connection with the fusiform bacillus, but nowhere as frequent as in the tonsil. The adenoids are not as favorable for the formation of masses of fusiform bacilli and spirilla.

He had come across an interesting specimen which had ideal conditions for the possible formation of such an abscess as was mentioned by Dr. Pollock—that is, the fusion of two clefts with a canal underneath the union, but in that instance there was no abscess in connection with the canal formation.

Dr. Samuel Salinger (closing the discussion on his paper) stated that the scope of his paper did not include the catarrhal form of nasopharyngitis which is associated with common colds in children. He wished to call attention only to the particular form of acute lacunar adenoiditis, and did not expect to touch upon the other types of adenoid infection, which is a large field and which would include Thorwaldt's disease, the later having no connection whatever with the acute type he considered.

As to the use of the nasopharyngoscope, if one is called to attend an acute condition in a patient's home, he is not likely to have the pharyngoscope with him. Secondly, in the presence of a large mass of mucopurulent material there is much difficulty in getting a good view. With the nasopharyngoscope one can get an excellent view of a limited area but not a general ensemble, and while the nasopharyngoscope has its place in some conditions, Dr. Salinger thought it was better if one could see the condition as it exists without the aid of mirrors or magnifying glasses, and in its relationship to the surrounding structures.

In regard to the enlargement of the posterior cervical glands he found this was very frequently mentioned in connection with inflammation of the adenoid. Many authorities consider this finding to be particularly pathognomonic of infections in the nasopharynx.

Dr. Robert Sonnenschein asked Dr. Davis if he did not think Dr. Salinger was correct in using the term "lacunar" instead of "follicular." Would not the same nomenclature be applied to the adenoid as to the tonsil? The actual "follicles" of the tonsil are lymphoid masses lying beneath the mucosa. Ordinarily when the term "follicular tonsillitis" is employed, it refers to infection of the crypts or lacunæ of the tonsil, hence the expression "follicular" is really a misnomer in these instances.

Dr. Davis, replying to the questions, thought the point of Dr. Sonnenschein was well taken. The follicles as they are seen in sections often show necrotic tissue and various inflammatory cells, and seem to be the part of the organ which exhibits the greatest degree of change.

Regarding the manifestation of disease, brought out by Dr. Lewy, the speaker thought one should consider "disease" as a relative term. He did not suppose anyone was really normal or absolutely free from some pathology. Bacteria are constantly passing into our interior, and we are constantly absorbing toxins, particularly through the gastrointestinal canal. This is evidenced by the fact that in the normal newborn child there are practically no plasma cells anywhere in the body, even in the tonsil or in the appendix. They do not appear until several days after birth when bacteria become prevalent in the tract. As the bacteria and toxins are absorbed the body defends itself in one way by inflammatory cells. In saying that many infections appear in the region of the ileocecal valve and in the region of the oropharynx he meant that the chief manifestations of disease appear in these two localities.

With reference to the diphtheria carriers, Dr. Davis believed the extirpation of the palatine tonsils would diminish the number of bacteria carriers but did not think it would do more than that. It might or might not cure a carrier, but would probably reduce the number of germs present.

JOINT MEETING OF THE CHICAGO MEDICAL AND CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETIES.

A joint meeting of the Chicago Medical and Chicago Laryngological and Otological Societies was held on Wednesday evening, November 9, 1921, with Dr. Robert Sonnenschein, President of the Chicago Laryngological and Otological Society, in the chair.

DR. M. A. GOLDSTEIN, St. Louis (by invitation), addressed the society on

"Problems of the Deaf."

ABSTRACT.

Not only the general medical profession but even otologists throughout the country know very little about the work that is being done in conjunction with the education of the deaf children. Chicago is peculiarly fortunate in its relation to the deaf child. It is the first city west of the Alleghanies that developed oral training for deaf children.

There are two methods of oral training of the deaf child, one of which is by sign language, in which signs, instead of speech, are used, and the other method is to teach children who have never heard the sounds of the human voice speech, even though it is artificially produced, and lip reading, so that they can come in contact with the rest of speaking humanity. The deaf child can be taught to speak and to come in contact with people by lip reading, a method which is used both for the child and the adult where hearing has been either congenitally or adventitiously lost

The public school system of Chicago has since 1896 shown the possibilities of oral training and speech training for deaf children instead of the sign language. Much of this good work has been brought about by the conjoint request of the parents of deaf children and of boards of education who feel that deaf children should have such opportunities as are offered normal children, in order that they may grow up to manhood and womanhood. It has been a long and hard struggle for the few who have interested themselves in these prob-

lems to actually convince other members in the profession that the opportunities for deaf children are great. Splendid progressive work throughout the country is being done, and otologists should help to shoulder the responsibilities which tend toward the development of deaf children. It is by the co-operation of the medical profession, and particularly the otologic and laryngologic members of the profession, that improvement in the education and development of methods for deaf children can be brought about. The sky has been cloudy for one-quarter of a century, but the dawn is coming, and there are specialists throughout the country who are beginning to show more than a passing interest in these problems of deaf children. There is a responsibility that medical men and otologists owe to deaf children, and the deaf child is as much entitled to a share of consideration as is the blind child, the normal child or the crippled child. Every child born deaf, who is not subnormal and has no mental defects, should be given the opportunity of being taught like normal children. Every congenitally deaf child can be taught fluent speech, as has been proven in all large metropolitan centers of the country. Deaf children, who have had six or seven years of training in their respective schools, are going to high schools and are sitting in the classroom with normal children and carrying on their work. For instance, there are three or four deaf children in Chicago who are doing that, one of whom is the scholarship pupil in his class. He is getting his lessons by lip reading. This is the opportunity the teacher and the otologist is creating for the deaf child in the future. The time is coming when teaching methods will become more perfected, when imperfections in speech will be corrected. Not all deaf children speak distinctly, nor do all doctors speak distinctly, but this should not be taken as a criterion.

Through the courtesies of the teachers of the several schools for the education of the deaf in Chicago, and to show what progress is being made and what is being done in actual practical work in the schoolroom, Dr. Goldstein called the classes one by one and had the teachers demonstrate what the deaf children could do by lip reading and other methods of oral training. There were classes representing the Parker Practice School, the Beidler School, and the Alexander Graham Bell

School. Demonstrations were given by the children through their teachers. Miss Plumb of the Parker Practice School and her pupils gave a demonstration of the first signs of speech. When Miss Plumb spoke the words thumb, shoe, eye, elbow, nose, etc., the pupils pointed respectively to their thumbs, shoes, eyes, elbows and noses. She then showed the first elements in speech bv lighting a candle and having the children blow it out by uttering the sounds P, O, sh, T, wh, and ah.

Dr. Goldstein said that musical instruments are used, besides the voice, in training children to learn rhythm, accent, and to appreciate pitch. In some schools this is being developed to a considerable degree. Demonstrations of rhythm work were given by Miss Smith of the Parker Practice School with her pupils, with Miss Jones at the piano. Music is used not only for rhythm but to educate tactile sense.

Miss Knox of the Alexander Graham Bell School gave demonstrations of what the pupils in the hard of hearing class could do by lip reading.

Miss Golden of the Beidler School, with her pupils, gave demonstrations in lip reading, and Miss Taylor of the Parker Practice School gave a demonstration of what her pupils could do in arithmetic by lip reading. These demonstrations showed that every congenitally deaf child can be trained to speak and can be trained to lip read to such a degree as to become a useful member of the community in which he or she lives.

DR. GEORGE E. SHAMBAUGH read a paper entitled

*"Problems of the Hard of Hearing and the Use of Electric Hearing Devices."

*See page 225.

MISS VALERIA D. McDERMOTT, executive secretary of the Chicago League for the Hard of Hearing, read a paper entitled

"Community Organization for the Deafened."

ABSTRACT.

The work of the league begins at the place where that of the otologist ends. Its primary purpose is to ameliorate the condition of the deafened. And what is the condition of the

deafened? This question can best be answered by giving a typical illustration:

With all hopes of cure removed, unable to enjoy or take part in family or friendly conversations or hear a concert, lecture, theater or church service, or earn a livelihood in a profession or occupation in which they have spent years of preparation or service, they come to the league. A hopeless condition, it would seem. But it is not, for the league has yet to encounter a case that it could not help. In many instances it has transformed discouraged, listless, purposeless, unproductive individuals into happy, self supporting members of the community.

How is this accomplished? Not by an experiment, but by an organization and a plan worked out by those who had a psychologic and sympathetic understanding of the problems of deafness—the deafened themselves. In this respect it differs from all other organizations working with handicapped groups and also explains its success as a growing and spreading movement, for there are now twenty-two cities with like organizations doing a similar work for the hard of hearing.

The first assistance given to those who come to the league is to offer a substitute for their impaired or lost faculty, sight for hearing—teaching them to read the lips. Then, unconsciously, through association with others who have met the same handicap and overcome it, confidence is inspired and self pity vanishes.

There are many things that persons with defective hearing can do. There are vocations and professions just as interesting as those that have to be abandoned. This is the psychologic attitude that the league creates in relation to employment, and gradually those who have in their own minds despaired of being able to earn a living become interested, begin to inquire, want to retrain or will try the position advised.

From April 1, 1920, to March 31, 1921, 6,979 persons came to the league; 2,661 men and women attended the lip reading classes; 1,382 lessons were given to twelve ex-service men sent to the league by the Federal Board for Vocational Education. A lip reading class for adults was established in the Lowell School, Oak Park, Ill. Three scholarships in lip reading were awarded, one to a shut-in girl and two to young women employed.

Employment was found for applicants in the following lines of work: Housework, typing, bookkeeping, filing, billing, addressing, cashier, commercial art, press clipping, accessioning, assembling, printing, proofreading and gardening. A study of professions and occupations that the hard of hearing can follow was begun and is being continued. Vocational counsel was given, and as a result a number of persons are pursuing courses of study. Three ex-service men are receiving training in industrial chemistry, mechanical dentistry and cabinet making. Two women finished courses and were placed in positions in the filing department. One man is taking a course in watchmaking, and two women, a teacher and a trained nurse, were advised to pursue courses, one in arts and crafts, and the other in microscopic analysis. These two women were given scholarship loans by the league, making it possible for them to take the training advised.

This year the league plans to project its activities into the community, becoming more a public institution and less a private agency. The establishment of a community house for the deafened similar to those already established in Philadelphia, Boston, Toledo and San Francisco is now under consideration. The employment service of the league is to be extended, for herein organizations for the hard of hearing can render the greatest assistance. The third feature of the program takes the league into the field of prevention—the establishment and maintenance by the league, in cooperation with the Chicago Department of Health, of an ear clinic in the public schools.

It is just as important, and possibly more so, that the children's ears receive proper examination and treatment, as their eyes or teeth, although there is only one city in the United States—Rochester, with its eminent Dr. Franklin W. Bock—that has gone into this field and done a large and constructive work. The matter of establishing a clinic is now before the executive committee, and the details are not entirely worked out, so that it can only be referred to at this time as a possibility for greater service, and a measure taken for the prevention of deafness in early life.

DISCUSSION.

Miss Mary McCowan, teacher of the deaf in Chicago, stated that 38 years ago she came to this city and established a small experimental school for teaching the deaf. In those days, when they spoke about music and rhythm for deaf children, they were laughed at, and when they talked about speech for deaf children they were considered impostors. She rejoiced to have lived to see the day when it was an accepted fact that deaf children could be taught to speak, and it was our duty to see that they were taught to speak.

Dr. Frank G. Brunner, Director of Special Education, Public Schools of Chicago, said the teachers of this city would welcome any suggestions from members of the medical profession in reference to better methods of teaching the deaf. He said we were just at the threshold in the teaching of the deaf, and phenomenal progress might be expected in the future in comparison with what had already been done. Any amount of time was worth spending if we could put children in possession of speech and make it possible for them to associate and converse with normal individuals in the community. His experience had been that physicians, and sometimes even otologists, did not seem to appreciate the necessity of getting deaf children into schools for the deaf at an early age. Physicians not infrequently kept these children out of a school for the deaf until they had begun to lose their speech or until their speech had become so imperfect that the teacher had to spend considerable time in correcting bad habits of speech before beginning to develop anything like intelligible speech. It is not necessary, as some had supposed, to consult politicians in order to get such children into schools for the deaf. Deaf children are admitted to the schools for the deaf when four years of age, but he did not think it was wise to admit them younger than this.

Miss Gertrude Torrey, principal of the Chicago School of Lip Reading, said the members of the society had seen an excellent demonstration of what children could accomplish with lip reading. A great many people become deaf after reaching adult age, and they need lip reading as much as the children. Probably all doctors who recommended lip reading thought it was only the young people who needed this education, but most important of all was the change of attitude of mind which came from lip reading, and all doctors ought to think of this in recommending lip reading, not only to people who were deaf but to those who were becoming deaf. Almost all people who were a little deaf were in a despondent frame of mind and thought there was nothing ahead of them. When such people took up lip reading they felt more hopeful. After seeing what other people could accomplish by lip reading, gradually they came to realize that they themselves could become very good lip readers.

DR. NORVAL H. PIERCE stated that not long since, he examined the inmates of a large deaf and dumb asylum and found that at least 10 per cent of those he examined could hear more or less, and yet those children were living a life of silence and most of them communicating with one another by means of the sign language. Strange as it might seem, these children were admitted to this institution without the slightest scientific examination.

In looking over the application blanks admitting these children, the cause of deafness and the kind of deafness were filled in by the parents, so that it was not an unusual thing to read that the deafness was ascribed to causes which might provoke a smile if the matter were not so tragic.

State institutions should keep in close touch with well informed otologists, and while it was true that otologists had not been as much interested as they might have been in this subject of training and education of the deaf, he was inclined to think that teachers of the deaf had not been quite as much interested in otologists as they might have been. There was no doubt that these two component parts had in this country developed along separate lines. This could not be said of Germany, where a great advance in the education of the deaf by the oral method had been made, the work being furthered by professional otologists. He believed it would be a good thing if from this meeting a closer communion could be established between otologists and teachers of the deaf.

He wondered if all of the children who had given demonstrations tonight had been thoroughly examined by competent otologists. If they had not been, the system of education might be materially improved by the cooperation of otologists and teachers of the deaf.

Dr. Joseph C. Beck said that one of the greatest accomplishments in connection with Dr. Goldstein's work with deaf children was to make teachers in his institution who could carry on the work. It was such splendid demonstrations as Dr. Goldstein had given that stimulated otologists to become more and more enthusiastic to do better work. One difficulty lay in not being able to teach deaf children privately, as was required by some parents, particularly the better class of children, who in addition to attending a school for the deaf wanted private tutors. It had been difficult at times for the teachers to devote the necessary amount of time to give these children private instruction.

In regard to the humming, buzzing devices referred to by Dr. Shambaugh, the speaker called attention to a device known as Rice's oscillator, which was supposed to greatly aid the deafened. No differentiation of cases was made, but this oscillator was used in all kinds of cases. In cases in which a diagnosis of fixation of the stapes was made, he was anxious to see if these people could be made to hear again with this oscillator.

In the East at the present time there was a great deal of discussion going on with regard to the use of the X-ray in the treatment of deaf individuals.

Dr. Elmer L. Kenyon stated that in a certain school in Chicago, largely attended, there had been up to within a few weeks a brother and sister attending, aged 13 and 15 years respectively, whose hearing was so impaired they could not possibly hear what was going on in the classroom, and yet had been attending this school (not a public school) year after year. These children should have been properly educated in deaf schools. Why they should have been continued year after year in a school for normal children, accomplishing practically nothing in the way of their own education, he could not understand. Nothing could point with more emphasis to the need of adequate medical inspection in the schools than such serious mishandling of deaf children.

Dr. Kenyon cited the case of a man, 24 years of age, who could not talk understandingly, could not hear well, could not read and who seemingly lacked in normality of intellect. Examination showed that he could hear loud talking on contact with the ear. His mind was normal, considering the handicaps he had gone through. He could not read more than perhaps every tenth word in a child's book. He had been deafened when he was a child and had had practically nothing done for him. Finding the public school almost useless, he had, after three years of attendance, been taken out, and afterwards had simply been allowed to drift, uneducated, into manhood. Meetings like the present, spreading knowledge of such problems, should eliminate such tragedies.

Dr. M. A. Goldstein, St. Louis, in closing the discussion, said it required a tremendous amount of patience, forbearance, originality and individuality on the part of the teacher of the deaf child. There was no better work being done for the deaf in any of our metropolitan cities than in Chicago. If otologists, friends of the deaf, and representatives of the League for the Hard of Hearing fully realized what this kind of training would do, he believed Chicago would be in the vanguard of this work for many, many years.

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XIX.

FATALITIES FOLLOWING OPERATIONS UPON THE NOSE AND THROAT NOT DEPENDENT UPON ANESTHESIA—A STUDY OF THREE HUNDRED AND THIRTY-TWO HITHERTO UNREPORTED CASES.*

BY HANAU W. LOEB, M. D.,

ST. LOUIS.

The development of the operative field of laryngology has been one of the most startling advances of medical science. From being an occasional operator, the laryngologist has become an accomplished surgeon with an enormous operative material—far beyond that of any other surgical specialist.

The work has become so attractive—or, perhaps, so remunerative—that there are today five thousand physicians in

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*Read before the American Laryngological, Rhinological and Otological Society May 4, 1922.

By resolution of the Society, the writer has been asked to continue and extend these studies. Accordingly, these cases will be included in those to be reported in the paper to be presented next year.

America, according to the American Medical Directory, who profess to be qualified to practice this specialty, one out of every thirty practitioners in this country.

In this large body all are not well trained, all are not skillful, all are not progressive, all are not mentally alert, all are not conscientious. Be this as it may, they have an important bearing on the well being of the people of today.

Considering the tremendous operative activity of laryngology, it is important to gain some estimate of its mortality risk. The fatalities must be excessively small in percentage, considering the number of operations. The meagerness of the literature on the subject shows this; and yet the published reports are sufficient to justify a more searching inquiry with the view of assembling reports of fatalities which have not been published.

It has been deemed advisable to exclude in this paper the anesthesia fatalities which would bear approximately the same relation to the total number of cases as in other operations of equal grade, with the slightly added danger which surgical work about the air passages would entail.

Inasmuch as the writer has no desire in this paper to touch upon cases in which the disease processes or the operative procedures were of themselves of a serious character, it was decided to limit the study to operations upon the nose and throat, as follows:

1. All intranasal operations on the walls of the nasal cavities and paranasal cavities, excluding pituitary operations and those performed for malignant disease, and

2. Tonsillectomy, adenoidectomy, incision for peritonsillar abscess. Endoscopic operations upon the larynx, trachea, bronchi and esophagus are excluded in view of the small proportion which the endoscopists bear to the total number of laryngologists. Furthermore, a study of such fatalities belongs more appropriately to an active endoscopist.

The writer became interested in the subject after the occurrence of a fatality in his practice following a submucous resection.

REPORT OF CASE.

H. C. C., an attorney, aged 52, consulted me on February 25, 1921, complaining of slight pain over the left ear for some months, which had become somewhat worse lately, and which was more severe at night. His right ear had been deaf for a year, while in the left the hearing had just begun to diminish. Examination showed: Hearing: R., voice, 4 meters; whisper, 0.1; L., whisper, 4. Weber lateralized to the right. Rinne negative, right; positive, left. Nasal septum was greatly deflected to the right superiorly and to the left inferiorly. Diagnosis of chronic catarrhal otitis media was made.

Inflation improved his hearing very much and reduced the pain. It was felt wise to perform a submucous resection upon the septum in order to give him ample air for audition and for nasal respiration. Operation was accordingly performed on April 1, 1921.

He left the hospital feeling very comfortable on the next day. On April 4th he returned to the hospital on account of severe hemorrhage. The intern plugged his nose with gauze, which was allowed to remain until the following day when I removed it. In the afternoon he had a second severe hemorrhage, for which it was plugged again with gauze by the intern. This was removed that evening without a recurrence of any hemorrhage except a very slight oozing. On the day after his return to the hospital he developed acute tonsillitis on both sides. The patient was feeling very comfortable until three days later, when he was taken with pain in the occiput, marked deafness and an abscess in his left ear.

On the following day he was delirious, absolutely deaf, with an abscess in right ear but with no rigidity of neck. The head could then be rotated in all directions. No Kernig present. Examination showed bulging drum and hemorrhagic discharge from left ear. Paracentesis was performed, after which there was a copious purulent hemorrhagic discharge. During the evening manifest symptoms of meningitis appeared, and signs of facial paralysis, with neck rigidity and definite Kernig. He died that evening.

Autopsy showed middle ear on both sides filled with thin purulent material; purulent meningitis marked over the petrous bone on both sides; tuberculosis, left lung; pneumothorax, left side; arteriosclerosis; infectious thrombosis of liver; chronic interstitial nephritis.

There was no lesion over the cribriform plate on either side, and the sinuses were free from pus.

Only one other case falling within the scope of this paper came to the writer's observation through his practice.

This was a man whom I saw in consultation with a laryngologist who, in attempting to remove the superior turbinate bone, had fractured the cribriform plate. Meningitis developed within twenty-four hours and death ensued on the following day.

INVESTIGATION.

As has been already stated, the literature of the subject is exceedingly meager, being made up mainly of reports of isolated cases, with occasional autopsy protocols and with an attempt to explain the occurrence of death in the case reported. There has been very little constructive work done looking towards frequency of a fatal issue, and its reduction or elimination. The small number of cases reported militates against the expression of any conclusions which could be of much value.

The writer, therefore, undertook to assemble a larger number of cases, in the hope that a better understanding as to the frequency of such fatalities might be developed and that some conclusion of worth might be drawn. To this end a letter was sent to each of the five thousand practitioners of laryngology in this country, asking for reports of cases.

It is understood, of course, that such reports cannot have the weight of those which come from a single controlled clinical source. Factors of error are bound to creep in, depending upon the multiplicity of observers, the designed or unintentional coloring of the reports and much unavoidable looseness of observation. And yet there has been a surprising helpfulness manifested, which expresses the profound interest in the subject and the earnestness of the reporters themselves. Four hundred and sixty-seven answered that they had had no fatalities whatsoever. Two hundred and fifty made reports which this paper analyzes, and approximately 4,300 have thus far made no answer. As a result of this inquiry, I have reports of 332 fatalities, more or less complete and carefully collated to avoid duplication. When this large number is compared to the paucity of published cases, it is obvious from the very first that we have not given to the subject the importance or the seriousness it merits, nor have we had sufficient data to justify conclusions respecting it.

It is, of course, impossible to determine with positiveness the causes of death in all the reports assembled, because there has been no set formula to guide these, and because of the variant conditions under which the observations have been made.

Altogether thirty-two autopsies are included, but these in many instances have been made by untrained men. The protocols are, therefore, not always of use. However, in spite of the possibility of inaccuracies as outlined, the writer feels certain that something of value can be had by the classification which has been made.

The following grouping of the fatalities is made with the number reported in each group:

I.	Meningitis	125
II.	Hemorrhage	55
III.	General sepsis	20
IV.	Erysipelas	8
V.	Endocranium (excluding meningitis)	26
VI.	Respiratory tract	43
VII.	Heart	5
VIII.	Miscellaneous	20
IX.	Undetermined	30
	Total	332

MENINGITIS.

Meningitis forms by far the most common cause of death. Although in the vast majority of the reports it followed nasal operations, in one instance it was the cause of death following adenoidectomy. The operation in this child, aged nine months, was performed to relieve convulsions, death occurring twenty-four hours later. It is likely either that the meningitis was present before the operation or that the endocranium was injured during the procedure.

There were four tonsil operations in this category. In considering tonsillectomy, no distinction is made as to whether or not there was a coincident adenoid operation. In two of these, an acute suppurative otitis media intervened between the operation and the meningitis, a possibility that can be readily understood. As is well known, a postoperative otitis media suppurativa is commonly of a virulent type.

In one of the other cases the meningitis followed immediately after operation; in the other, ten days later, both rapidly succumbing to the disease. The causal relations in these two cannot so readily be explained.

Meningitis was the cause of death after 120 nasal operations as follows:

1. Intranasal frontal operation	16
2. Probing and irrigating frontal sinus	7
3. Ethmoid operation, with or without resection of the middle turbinate	39
4. Sphenoid operation	10
5. Maxillary operation	1
6. Resection of middle turbinates	15
7. Submucous resection	13
8. Removal of polypi	13
9. Combined sinus operation	1
10. Combined submucous and sinus operation	5
Total	120

This is certainly an appalling list, which cannot be lightly cast aside. The preponderance of fatalities is altogether on the side of the operations which come into relation with the lamina cribrosa. Perhaps the suggestion contained in the older books that the cribriform plate can be recognized by its hardness and resistance to the instruments may be somewhat responsible for the frequency of this fatal complication. In seventeen instances the reporters noted the occurrence of fracture into the cranial cavity, but there were doubtless many instances where this accident was overlooked.

Several reporters saw the cerebrospinal fluid escape during the operation. Another undertook to remove the superior turbinate, without disturbing the middle turbinate. Another stated that he removed the entire cribriform plate which was sequestrated. Quite a few used the Ballenger ethmoid exenteration knife but discarded it after the fatality. In some, plugging the nose too long or too frequently appeared to be a factor. In others, an acute otitis media suppurativa incident to the operation was the forerunner, as in my own case.

In most instances, meningeal symptoms appeared within forty-eight hours after the operation. Can this be explained on the theory of an infection without fracture into the cranial cavity? If intracranial infection can make its presence known so soon after operation in the absence of fracture, what prevents its more frequent occurrence in the multitude of operations which disturb the bacterial relations? Are we wrong in asserting that the nasal cavities are most resistant to infections? Considerations such as these force us to the conclusion that most of the cases occurring within the first twenty-four hours are due either to a fracture or to the lighting up of a latent meningitis.

Of the nine reported autopsies made on patients dying from meningitis, fracture was found in five in whom the symptoms appeared during the first twenty-four hours; in one with no specification as to onset, and there was no fracture in the two cases sectioned in whom the symptoms appeared after forty-eight hours.

1. Intranasal Frontal Sinus Operation.—Considering the relative infrequency of the intranasal frontal sinus operation as compared with other intranasal operations, sixteen deaths from meningitis (13.3 per cent of all the cases) are sufficient to show the dangerous possibilities of this operation and to warn those whose knowledge of the anatomy is not sufficient to justify their undertaking it.

These considerations may not apply to the removal and curettement of the ethmoid cells, but they are forceful in reference to the drainage of the frontal sinus in connection with any attempt to enlarge its canal or orifice. One operator reports two cases in which the operation was followed by meningitis within twenty-four hours, one dying two days later and the other three days later. Another reports a case almost the counterpart of the first of these. In another, the meningitis with unconsciousness supervened immediately after the operation with a temperature remaining above 105 until exitus three days later. In four, the symptoms appeared several days after the operation when the sinus condition had improved, justifying the opinion that there had been an infection without fracture. Another case is reported in which the operation was performed one week after the appearance of influenza, complicated by an acute frontal sinusitis. Two days later meningitis developed and the patient died three days later.

2. Probing and Irrigating the Frontal Sinus.—This operation which many consider entirely free from danger is responsible for seven deaths, large in proportion to the number of such procedures undertaken. One of them developed the disease in a few hours, two in twenty-four hours, one dying a day later, the other two days later. The disease appeared two days after operation in two of the cases, several weeks afterward in another, and in one case the symptoms may have appeared before the operation.

3. Ethmoid Operation With or Without Resection of the Middle Turbinate.—In more than one-third of the meningitis cases reported, this operation was specified as the cause. All types of operation are represented, and, according to the reports, it is impossible to determine which type is most dangerous, with the exception of the Ballenger knife, which appeared to be most culpable either by reason of its mechanical construction or from inexpertness in its employment.

As no operations in which ethmoid curettement or exenteration was combined with those upon the septum or other sinuses are included in this group, the ethmoid procedure may be accountable for more fatalities than are here stated.

In nine the meningeal symptoms appeared within a few hours; in ten others, within twenty-four hours; in five others, within forty-eight hours; in eight of the remaining they occurred later, while in seven the time was unspecified. The onset of the symptoms has an important bearing on determining whether fracture into the cranial cavity has resulted, for, as has already been suggested, it is hard to conceive of a meningitis of lymphogenous or hematogenous origin occurring within twenty-four hours after the infection pathway has been first opened.

Escape of cerebrospinal fluid at the time of operation occurred several times. One of these was followed immediately by meningitis; another, after two weeks. Packing for eight hours to control postoperative hemorrhage is reported to have been followed in forty-eight hours by a fatal meningitis. One of the patients was a baby two years old. In one instance the operator, a well known and most skillful laryngologist, after the onset of the meningitis, removed the cribriform plate in the hope of relieving the process. He found evidence of a chronic pachymeningitis.

4. Sphenoid Operation.—The sphenoid operation is looked upon by laryngologists as more hazardous than that upon any other sinus, and they have imparted this view in large measure to their patients; hence, laymen are more prepared for a fatal issue in this than in any other type of intranasal sinus operation. A more or less extensive ethmoid exenteration practically always constitutes a part of the sphenoid operation, and naturally either may serve as an etiologic factor of the meningitis. The reports do not differ materially from those upon the ethmoid operation. In one case the meningitis came on before night and death occurred within seventy hours.

5. Maxillary Operation.—Only one intranasal maxillary operation was followed by meningitis, following what appeared to be a simple procedure in a long standing process.

6. Resection of Middle Turbinate.—This has an unusually large toll—fifteen—for what is ordinarily considered a very simple affair. In only one was there a coincident operation performed, resection of the corresponding inferior turbinate. Three developed meningitis the first day, one died on the second day with symptoms of acute mania, and one followed middle ear abscess and meningitis, supervening upon the operation. The last was performed by a general practitioner. The reports of this group are very incomplete, consisting almost entirely of a statement of the operation and cause of death. In one, cutting adhesions between the turbinate and septum resulted in meningitis in twenty-four hours and death thirty-six hours later.

7. Submucous Resection of the Septum.—Two factors combine to influence a meningitis following submucous resection: The perpendicular plate of the ethmoid and the exposure of

the medial sides of the septal mucosa with exclusion of drainage brought about by holding the two exposed surfaces together. If there was much disposition to nasal infection in the nose, we should expect far more frequent expressions of that infection than we get from this operation. However, the fifteen cases here reported constitute a sufficiently large mortality rate, from meningitis at least, to be of much concern to operators. In most of the cases reported, in which the onset is specified, the meningeal symptoms did not appear until after the second day, substantiating the view that these cases are largely hematogenous or lymphogenous. In one, the symptoms developed the first day and the patient died on the next, the reporter being sure that a fracture was the cause. Autopsy revealed a puncture of the cribriform plate in another in which meningitis appeared soon after operation.

My own case, reported in this paper, comes under the category of secondary meningeal infection resulting from otitis media.

8. Removal of Polypi.—Although removal of polypi was one of the first nasal operative procedures to which our literature refers as a cause of meningitis and death, the thirteen cases here recorded will be considered a number far beyond the expectation of laryngologists. The natural inference would be that the forcible evulsion of the polypi with forceps must have caused a fracture into the cranial cavity or so close to it that infection was possible.

Several of the reports are exceedingly interesting. One patient became semistuporous within twenty-four hours. No autopsy was made but the operator thinks that a meningitis was developing at the time of the operation.

One surgeon removed a polyp from the superior meatus with a snare. He heard what he calls a rucking sound. Two days later the patient developed meningitis, which was soon followed by death. In another, an ethmoid exenteration had been done some time before; soon after the polypi were removed meningitis developed. The surgeon thinks that there was a necrosis of the cribriform plate which caused the acute meningitis by activation through the operative procedure. A patient immediately after the removal of several polypi from the nose fell into convulsions, and was taken with meningitis followed by death. In another, from whom some polypi had been removed, autopsy showed polypi extending into the sphenoid. In another who died of meningitis eight days after operation, autopsy showed the lamina cribrosa on both sides as thin as paper through which the polypi in the ethmoid were visible. There was a very thin fracture running across the right cribriform plate and an abscess in the frontal lobe.

9. Combined Sinus Operation.—A combined sphenoid, maxillary and ethmoid operation resulted in meningitis and death in one instance.

10. Combined Submucous and Sinus Operation.—The operations performed were submucous and bilateral ethmoid exenteration, three, in one of which there was fracture of the cribriform plate with escape of cerebrospinal fluid; submucous, exenteration of ethmoid and intranasal operation on the frontal; and submucous resection with exenteration of ethmoid and intranasal operation.

HEMORRHAGE.

Hemorrhage constitutes the next most important group and is in evidence in both the nose and throat operations, as follows:

1. Tonsillectomy (with or without coincident adenoidectomy)	43
2. Adenoidectomy alone	2
3. Incision of peritonsillar abscess	3
4. Submucous resection	2
5. Sinus operation	4
6. Resection inferior turbinate	1
Total	56

1. Tonsillectomy.—It is certain that the forty-three deaths from hemorrhage after tonsillectomy do not by any means constitute all the unreported cases in this country. On the contrary, I am convinced that there are many more yet to be uncovered.

An analysis of the records shows that in sixteen there was a lack of postoperative control—that is to say, the operation was performed in the office and the patient sent home; or the parents insisted on taking the patient away; or there was lack of observation whereby the bleeding might have been recognized; or the operator left the city; or the physician in charge was unfamiliar with methods of producing tonsil hemostasis. Most of these patients would have been spared under other circumstances.

Hemophilia, mainly unknown to the operator, is charged with seven of the fatalities; in one the operator discovered too late that the patient's brother had died from hemorrhage after tonsillectomy, the basis of which was hemophilia.

In another instance, familial hemophilia was discovered after the death of the patient.

A fatal result ensued in four cases in spite of thoroughgoing ligation of the vessels. The fatality was immediate in three cases. Persistent uncontrollable bleeding occurred twelve times and fatal secondary hemorrhage four times, one being after three weeks. In one of these cases the bleeding occurred five days after the operation, coming from the nasopharynx, the patient never having been seen by the operator after the operation.

There were two instances in which there was an anomalous branch of the carotid. In one this branch was found to leave the main trunk of the carotid at about the middle of the tonsil fossa, and the vessel was severed three inches from its origin. The section of the enucleated tonsil shows a portion of the arterial trunk of large caliber with two or three large branches, which supplied the upper portion of the capsule. Palpation of the tonsil, according to the operator, would have discovered this situation.

One patient was an idiot; another had extremely high blood pressure.

From these reports it is obvious that much can be done to reduce the mortality from tonsil hemorrhage.

2. Adenoidectomy.—Very little detail is given in these cases.

3. Incision of Peritonsillar Abscess.—Of the three cases, one was in a child in which the incision was followed by persistent bleeding, which continued for some days and for which treatment was of no avail. Of the second no detail is given. The third proved to be a hematoma, which had resulted from

erosion of the carotid or internal jugular, death being immediate.

4. Submucous Resection.—Two deaths from hemorrhage occurred after submucous resection; one was allowed to return home after three days, the physicians available not being expert enough to control it. As to the other, no details are given.

5. Sinus Operations.—Hemorrhage resulted in death following puncture of the maxillary sinus, the bleeding being from both the gums and nose. A resection of the nasomaxillary wall came to a fatal issue by reason of hemorrhage: another death after removal of polypi, and still another after ethmoid exenteration. In none of these cases was there anything to anticipate a fatal hemorrhage.

GENERAL SEPSIS.

The number of deaths from general sepsis is relatively small and is in keeping with the teaching that serious infections are rare in nose and throat operations, if we exclude meningitis. They include:

1. Intranasal maxillary operation	2
2. Submucous resection	1
3. Tonsillectomy	16
4. Incision peritonsillar abscess	1
Total	20

1. Intranasal Maxillary Operation.—In one of the cases symptoms of general sepsis developed three days after operation for an acute maxillary sinusitis, which resulted in death on the following day from general peritonitis, the appendix not being involved. In the second case, the death occurred two days after operation.

2. Submucous Resection.—In the single case reported, the fatal issue occurred eight days after operation. Packing had been retained for twenty-four hours.

3. Tonsillectomy.—There is a growing feeling that sepsis is more likely to occur under local anesthesia than under general. This is a measure confirmed by the reports which show that of those in which the anesthesia is specified, seven were local and two general. Localized swelling is characteristic of

the majority, tracheotomy, though without avail, being required in two. In one the pharyngeal tissues were found infected with colon bacillus; in another peritonsillar abscess developed; in another, chill, 106 temperature and abscess of the hip; another showed by cultures that there was an attenuated diphtheria infection.

4. Incision Peritonsillar Abscess.—It cannot be determined whether the general sepsis reported was due to the operation or to the process itself.

ERYSIPELAS.

The operator is accustomed to look upon erysipelas with more chagrin than any other complication unless it be preoperative. The grouping is as follows:

1. Submucous resection	4
2. Sinus operations	2
3. Resection middle turbinate	2
Total	8

1. Submucous Resection.—In one of the submucous cases the middle turbinate was also resected. One showed symptoms one day after operation, the others much later.

2. Sinus Operation.—Included in the sinus operations were one of removal of nasal polypi by snare, in which erysipelas developed on the second day, and one in which erysipelas was present at the time of the intranasal maxillary operation.

3. Resection of Middle Turbinate.—One of the erysipelas fatalities following resection of the middle turbinate was in an old man with a chronic nephritis; the other patient bled considerably, requiring a gauze plug which was not removed until the following day.

ENDOCRANIUM, EXCLUDING MENINGITIS.

This includes the following:

1. Brain abscess	5
2. Cavernous sinus thrombosis	9
3. Cerebral embolus or hemorrhage	12
Total	26

In the absence of autopsy the diagnosis of brain abscess and of cerebral embolus or hemorrhage must be more or less doubtful, but in the grouping the opinion of the operator is accepted.

1. Brain Abscess.—In one the diagnosis was confirmed by autopsy, in another by operation. In two, the operation performed was removal of polypi. One of these developed a frontal lobe abscess by extension from subperiosteal and subdural abscess. The other, two days after the operation, became drowsy with slight elevation of temperature and pulse rate from 50 to 70. He died in four weeks. One case resulted from retention of gauze after an ethmoid operation. Another had ear abscess a week after operation from which the brain abscess resulted.

2. Cavernous Sinus Thrombosis.—This extraordinary complication is probably due to a hematogenous infection which finds its counterpart in those cases of furunculosis and similar processes affecting the skin of the external nose, vestibule and lip. Altogether there were nine cases, divided as follows:

Tonsillectomy	3
Sinus operations	2
Incision peritonsillar abscess	1
Resection inferior turbinate	2
Series of nose operations	1

Two of the tonsillectomies were followed very soon by the symptoms, which developed very rapidly. The other was that of a young soldier who had had tonsillitis two weeks before. The operation was performed under local anesthesia without incident. On the fourth day he had a slight elevation of temperature, and on the following day there was beginning exophthalmos with slight blurring of the disc on the left side. Symptoms rapidly increased until exitus on the seventh day after the operation. Diagnosis was confirmed by autopsy. The sinus operations were puncture of the antrum followed by orbital cellulitis and exenteration of the ethmoid.

There was nothing extraordinary in the incision of the peritonsillar abscess, except the unusual character of the complication. In one of the cases of resection of inferior turbinate, an adenoidectomy was done at the same time; in the other, the operation was bilateral. In the last case, three or four unspecified nasal operations had been performed at intervals of two days.

3. Cerebral Embolus or Hemorrhage.—This complication is represented in the fatalities thirteen times:

Tonsillectomy	7
Incision peritonsillar abscess	3
Nose operations	3

Of the tonsillectomy cases, three were in patients over forty and appear to have been due to accompanying conditions. As to the other four, a reporter describes one as being some brain condition, probably thrombus; another is stated to have walked to the bathroom contrary to orders and succumbed after showing localized paralyses; another had hemiplegia from embolus of the right cerebral; the fourth died suddenly while sitting up in bed.

The three cases of incision of peritonsillar abscess, are all reported by the same observer, who may have overlooked other possible causes.

The two nasal operations were upon patients fifty-six and fifty-eight years of age. One, upon whom a submucous resection was made, had been a hard drinker with a blood pressure of 230; the other died suddenly when he was put back to bed after an ethmoid exenteration.

RESPIRATORY TRACT.

The fatalities coming under this heading are classified as follows:

1. Foreign body	5
2. Edema larynx and pharynx	6
3. Pulmonary edema	1
4. Pneumonia	17
5. Pulmonary embolus and abscess	14
Total	43

The operations concerned with fatal complications involving the respiratory tract are as follows:

Nose operations	10
Tonsillectomy	31
Adenectomy	1
Incision peritonsillar abscess	1
Total	43

1. Foreign Body.—The five reports are as follows:

A submucous resection was performed under ether and the nose plugged with finger cot stuffed with gauze, which was inhaled and followed by death, the cause being unknown until autopsy showed the gauze stuffed finger cot in the primary bronchus.

A child died on the table after tonsillectomy from asphyxia due to clotted blood in the bronchi.

A very fat short necked colored woman died after incision of peritonsillar abscess from pus entering the larynx and causing suffocation.

A tooth was dislodged during tonsillectomy under ether, subsequently causing death from inflammatory reaction.

A boy before tonsillectomy under chloroform had eaten a large amount of beans. Immediately after the operation he vomited about a pint of beans, some of which were inhaled and caused death from suffocation.

2. Edema of the Larynx and Pharynx.—These cases may all have been septic, though some were exceedingly rapid. In one the edema was so rapid and severe that the patient died within twelve hours after the operation; another for whom tracheotomy was done within twelve hours survived two days. One died two weeks after operation, and two others in one week; still another died of edema glottidis one month after operation.

3. Pulmonary Edema.—This was found associated with a miliary tuberculosis on autopsy after a death from tonsillectomy.

4. Pneumonia.—When pneumonia follows an ether operation it is usually assumed that the anesthetic is responsible. In practically all of the cases here reported the anesthetic

etiology may be discarded. One was due to the inspiration of a piece of cartilage in a submucous resection under ether, causing foreign body pneumonia; two others were septic pneumonia from tonsillectomy, one under gas oxygen and one local; another after an intranasal antrum and frontal operation under local anesthesia developed septic pneumonia. The complication followed a curettement of the ethmoid exenteration and also an intranasal maxillary operation. Among the conditions blamed for pneumonia after tonsillectomy may be mentioned aspiration, influenza, recurring bronchopneumonia, a coincident peritonsillar abscess. All of these cases but one were under local anesthesia.

5. Pulmonary Embolus and Abscess.—Pulmonary abscess has been held as due to the general anesthesia, but the investigations of Cutler and Hunt (Arch. Int. Med., Chicago—1922—XXIX—449) show that it usually results from embolus. Hence it belongs under the category of this paper and not under that of anesthesia. Many more cases of pulmonary abscess will undoubtedly be discovered if the investigation is extended. All the cases of pulmonary abscess following tonsillectomy were general except one which was local and one not specified. In the reported exception the complication followed a resection of the inferior turbinate under general anesthesia. One reporter had three cases. Another gives the record of autopsy, which showed suppuration of the posterior ethmoid and sphenoid present. Of the four cases of pulmonary embolism, two were confirmed by autopsy.

HEART.

The five cases of heart cause of death comprised the following: Angina pectoris, after tonsillectomy; dilatation of the heart after removal of polypi; cardiac asthenia after tonsillectomy in a patient with grave heart lesions; cardiac infarct after incision of peritonsillar abscess and rupture of heart confirmed by autopsy in a submucous resection.

MISCELLANEOUS.

This list includes the following:

1. Acidosis	1
2. Delirium tremens	1
3. Dilatation of the stomach	1

4.	Diphtheria	1
5.	Epilepsy	1
6.	Hyperthyroidism	2
7.	Orbit injury	1
8.	Poisoning	1
9.	Scarlet fever	3
10.	Shock	1
11.	Status lymphaticus	5
12.	Uremic coma	1
13.	Uterine hemorrhage	1
	Total	20

The acidosis fatality occurred in a diabetic upon whom a local tonsillectomy was performed.

The delirium tremens followed a sphenoid operation.

Acute dilatation of the stomach resulted, according to the reporter, from a heavy meal a half hour before an ethmoid exenteration.

Diphtheria caused the fatality after a tonsillectomy in a man fifty-five years of age.

The operator had no knowledge of previous occurrence of epileptic seizures in a patient who died in an attack after a resection of both turbinates.

Hyperthyroidism unrecognized previous to operation was held responsible for two deaths from tonsillectomy.

An inexpert operator pushed an instrument into the orbit in doing a submucous resection.

Another injected an overdose of adrenalin by mistake instead of novocain before a tonsillectomy, causing death.

Three cases developed scarlet fever after tonsillectomy and died.

Shock was held responsible for a fatal issue after tonsillectomy in a patient with goiter.

Four tonsillectomies and one ethmoid exenteration succumbed to status lymphaticus, in which, of course, the anesthetic relation must be considered.

Uremic coma caused the death of a man suffering from an intense nephritis upon whom a submucous resection, nasomaxillary operation and an ethmoid exenteration had been done.

A woman with a high blood pressure expired suddenly from a profuse uterine hemorrhage sixteen hours after a tonsillectomy under general anesthesia.

UNDETERMINED.

In thirty cases no cause was determinable, either by the physician in charge or by the writer from the reports submitted. The operative procedures concerned were the following:

1. Tonsillectomy	16
2. Adenectomy alone	1
3. Exenteration ethmoid, with or without removal of polypi	4
4. Puncture of nasomaxillary wall, irrigation of the sinus	5
5. Submucous	1
6. Resection middle turbinate	2
7. Combined tonsillectomy and submucous resection	1
Total	30

The interesting cases in this group are those in which there was puncture of the nasomaxillary wall and irrigation of the sinus. They may well be compared with the sixteen already published and discussed at great length by Gording and Grove. There are doubtless some cases of status lymphaticus and unrecognized hemorrhage included in the group of undetermined causes.

TABULATION.

The table shows the cause of death in each type of operation. It is to be noted that tonsillectomy heads the list with 126, ethmoid exenteration being next with 46. The throat operations number 142, the nose operations 190.

TABLE SHOWING OPERATIONS AND CAUSES OF DEATH.

	Frontal	Ethmoid	Sphenoid	Maxillary	Resection Middle Turbinate	Resection Inferior Turbinate	Submucous Resection	Removal Polypi	Combined Sinus Operations	Combined Submucous and Sinus Operations	Combined Submucous and Resection of Either or Both Turbinates	Tonsillectomy	Adenoidectomy	Incision Peritonsillar Abscess	Total
Meningitis	23	35	14	1	15	....	13	13	1	5	....	4	1	....	125
Hemorrhage	....	1	....	2	....	1	2	1	....	....	....	43	2	3	55
General Sepsis	....	....	....	2	....	....	1	....	....	....	....	16	....	1	20
Erysipelas	....	....	....	1	2	....	2	1	....	....	2	....	....	....	8
Endocranium	....	4	....	1	....	2	1	2	....	10	1	....	1	4	26
Respiratory Tract	....	1	....	1	1	1	3	....	1	....	2	31	1	1	43
Heart	....	....	....	....	....	....	1	1	....	....	....	2	....	1	5
Miscellaneous	....	3	1	....	1	....	1	....	....	1	....	13	....	....	20
Undetermined	....	2	....	5	2	....	1	2	....	....	....	17	1	....	30
Total	23	46	15	13	21	4	25	20	2	16	5	126	6	10	332

REFLECTIONS.

Anyone who studies these reports, as the writer has, must be profoundly affected by them, particularly if he is at all sensitive to the unhappy experience which confronts the physician when one of his charges unexpectedly succumbs to an operation.

With this feeling I have set down what I term reflections, preferring to call them after this somewhat indecisive term rather than to hold them as well grounded conclusions.

They are presented in the hope that they may in some measure, if ever so slight, serve to reduce preventable fatalities after operations on the nose and throat.

I. AS TO THE PHYSICIAN.

1. Knowledge of the anatomy of the nose and throat cannot be learned by operating on the living patient—and yet, how many undertake to acquire it by surgery rather than by arduous study in the dissecting room.

2. To operate without this requisite knowledge is impossible for a man of conscience, not to mention the hazard to the patient.

II. AS TO THE PATIENT.

1. The patient himself, and not the disease or the name of the disease should be studied.

2. Is he a proper subject for the operation? Is he too young or too old? How are his arteries, his heart, his lungs, his blood and his kidneys? Has he tuberculosis, syphilis, diabetes, carcinoma or some eventually fatal chronic process? Is the operation likely to benefit him or is it worth while to operate upon him, considering his circumstances and environment, whatever such an operation may do for anyone else? These are potent questions of concern to the patient.

3. What might be termed the casuistry of operative indications is a good thing to study, for patients are not all alike; their response to operative intervention varies and the results, good and bad, cannot always be the same.

4. A good internist is a powerful help in the estimation

of the patient's reaction. He has saved and will save many a life for the surgeon.

5. Under all circumstances where it is possible, the patient should be hospitalized. We can count many indeed, from the reports, who could have been saved if the operation had been performed in a properly equipped hospital.

III. AS TO THE OPERATION.

1. Rigid attention to asepsis is essential, not by washing the field with antiseptic solutions or by applying iodin, but by operating with an aseptic organization such as would be used in any other important surgical operation. The nose tolerates its own bacterial flora without much reaction but not extrinsic bacteria.

2. The sitting position is bad for nose operations—the patient's head is too easily moved; fainting is common; toxic symptoms from cocain occur readily; the physician's arms are in an awkward position, and instrument control is not always secure.

The semirecumbent position is far better. The patient need not be held; the range of head movement is greatly restricted, fainting and cocain intoxication are uncommon, the surgeon has better control over the operative field.

3. It is better to take out too little than too much—even if a second operation be required. There would have been less fractures of the cribriform plate if all of the operators had followed this rule.

4. Multiple unrelated operations are an abomination—what is gained on the one hand is more than lost by the increased danger, the prolonged healing, the added complications. Furthermore, when untoward symptoms appear, who can tell which operation is responsible for them?

IV. AS TO THE POSTOPERATIVE CONTROL.

1. To operate and then to consign the patient to inexpert hands can have no justification. Many of the fatalities here recorded tell the tale of such practice, whether the surgeon leaves the patient, or the patient returns to his home without proper supervision.

2. The nurse or the mother or the attendant, whoever it may be, must be alert, particularly in caring for those who cannot explain their symptoms.

3. A better surgeon is required to supervise the after-treatment than to perform the operation.

V. AS TO THE FATALITIES.

1. Mankind has use for everything—even for adversity. Every fatality should call for an autopsy, carefully made and carefully recorded and then published, for the best way to avoid fatalities is to study those that occur.

2. The study and analysis of well kept histories and well planned autopsy protocols will bring about definite conclusions which will be of the utmost value in reducing the number of fatalities for which the less serious operations on the nose and throat are responsible.

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XX.

OPTIC NERVE AND ACCESSORY SINUSES.

By Prof. J. van der Hoeve,

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Mankind is susceptible to inflammations of the nose. Every human being in his turn suffers from catarrh of the nose, some very often, others less frequently, but nobody is exempt from this trouble. Every time we have it, the inflammation may spread to the accessory sinus of the nose.

If we consider how very close and intimate the relation of these cavities is to the orbit and optic nerve, it seems almost a wonder that there are still some who have never had eye disease of nasal origin.

If out of the huge mass of eye diseases attributed to sinus affection we assemble those which concern the optic nerve, we must fix our attention upon several points.

1. The diagnosis of the optic nerve disease.
2. The diagnosis of the sinus affection.
3. The relations between these diseases.
4. The treatment.

THE DIAGNOSIS OF THE OPTIC NERVE DISEASES.

This diagnosis is less or more difficult according to the kind of nerve disease, which may be: choked disc, papillitis, atrophy and neuritis retrobulbaris Choked disc and papillitis, though it is not always easy to differentiate one from the other, are relatively easy to recognize, because the ophthalmoscope reveals in the very beginning that something is amiss with the optic nerve.

The diagnosis of the atrophy causes more difficulty, at least in the beginning, but the greatest difficulty is caused by the most frequent one, retrobulbar neuritis. The patients sometimes complain of pain behind the eyes in the depth of the orbit especially in movements of the eye. The ophthalmoscope is only of little benefit; in the beginning we see either nothing abnormal or a little hyperemia of the disc, dark and greatly filled veins, until at last the characteristic pallor of the temporal quadrant of the disc appears. But then it is too late, the affection which could probably be cured before, is usually irreparable, therefore we must look for symptoms which enable us to recognize the affection in the very beginning when the treatment may be more successful.

It is possible that in the future the examination of the light perception will help us, but this has not yet been studied enough to be of much value and the best indices are given us by the examination of the visual field. Here we find sometimes as atypical symptoms concentric contraction of the visual field or annular scotoma and as characteristic signs: central scotoma and enlargement of the blind spot.

We do not at present know why the first loss of function appears in the peripapillar and central fibers; we have only to accept the fact.

We know that both scotoma begin as relative scotomata for colors only, whereas later the white light is also not observed. As the scotomata enlarge, they may unite and cause in this way the well known oval scotoma, including and surrounding the fixation point as well as the blind spot.

As a rule the enlargement of the blind spot appears first: if only one of the scotomata appears, it may enlarge until it invades the region of the other and causes the same oval scotoma, including blind spot and fixation point.

If we find this scotoma, we cannot say in which way it was formed but only this that the peripapillar fibers and the central fibers are affected.

Of course when we find one of these symptoms we must exclude the possibility of another origin, for the central scotoma may be caused by degeneration or hemorrhage in the macula, etc., the peripapillar scotoma by high myopia, medullar fibers, hysteria, etc.

Both symptoms are very important and we know with certainty that they can be caused by sinus affections, for I know of different cases of enlargement of the blind spot, as well as of central scotoma, where the scotoma decreased or vanished by treatment of the sinus affection and came back by every recurrence of the sinus diease.

We find the optic nerve affection much more frequent in diseases of the posterior than of the anterior sinuses, in which they are very rare.

Important as these symptoms are, we must be well aware that they are not symptoms of sinus affection but only of affection of the optic nerve.

They show that the optic nerve is diseased but they cannot teach us anything of the origin of this affection and we must always consider that every retrobulbar neuritis may begin with these symptoms, whether it is caused by multiple sclerosis, by tuberculosis, syphilis, dental infection, rheumatism or from a nasal affection.

So we arrive at our first conclusion:

The ophthalmologist has in the eye no sign to distinguish the origin of a retrobulbar neuritis.

DIAGNOSIS OF THE SINUS DISEASE.

Every rhinologist will be convinced that the diagnosis of an existing sinus disease, especially of a posterior sinus disease, is not always easy, we may say, not always possible.

So we find the following in Dan. MacKenzie's Diseases of the Throat, Nose and Ear, edited in 1920:

"The absence of any sign of disease inside the nose is not sufficient to exclude sinusitis from the diagnosis."

With the purely rhinologic methods of examination we are often not able to diagnosticate an existing sinus affection; happily we have support in the examination with Roentgen rays. We can photograph the sinus in different directions, occipitofrontal, when we wish to compare both frontal sinus, bitemporal and occipitocaudal, according to Pfeiffer's method, to compare the both sphenoidal sinus.

For the ethmoidal sinus I always think the method of Rhese to be the best.

The patient lies down on his tuber frontale, nose and ansa jugularis and the tube is put above the protuberantia occipitalis. Then we get in a normal person a photograph like this figure; you see the frontal sinus, the orbit and the region of the ethmoid; in this method we should always compare both sides.

For the ophthalmologist it is of great interest that we can get in this way an excellent image of the foramen opticum and the fissura orbitalis superior, so that we can find out if something is amiss with the foramen opticum. In the various photographs the foramen appears as a nice round hole.

We photographed many persons with fractures of the skull-bones and opticus atrophy in this way to determine if we could find a cause for the atrophy. In this are seen the callous on the base line of the skull and a foramen opticum which has an hiatus at the upperside and a fissure running from the foramen opticum in the skull. Here an underbroken base line and a callous on the foramen opticum. The base line can also be disturbed by other processes, for instance by a gumma, as in this case of Sonnenkalb. Here you see a foramen opticum which has quite another shape, after a skull traumatism. I use this method also to see whether the foramen opticum is free in patients with towerskull. It is said that the opticus affection in towerskull is caused by a deformity of the canalicus opticus, and Schloffer even operates on this theory to free the optic nerve in the canal. If I find a normal foramen I do not believe the canal can be much deformed, in fact, I have found a normal foramen opticum even in cases in which there was marked towerskull with choked discs. Care must be taken not to misinterpret a picture the result of wrong position.

Rhese's method demonstrates affections of the sinus frontalis as can be seen in this case of osteoma which overshadows the foramen opticum, but the great advantage of the method lies in the examination of the ethmoid, as in this case of obscuration of the ethmoid, in posterior ethmoiditis.

How important this method can be may be shown by the following case:

A physician had neuritis retrobulbaris and choroiditis; he was operated on for sinus disease several times by one of the best known professors of rhinology, until the professor told him, that though he did believe that he had not opened all affected ethmoidal cells he did not dare to go farther.

When I saw this man, I found a huge enlargement of the blind spot especially for colors and hyperemic disc. I informed him of this and advised him to have his nose treated again. The rhinologist, however, thought him to be a neurasthenic and did not wish to operate any more. The man went to Dr. De Kleyn in Utrecht. Here Dr. Stenvers made a radiogram. The picture is shown here, and you see the whole ethmoidal region is clear except a rectangular spot close to the foramen opticum and fissura orbitalis superior. De Kleyn opened at that spot some posterior ethmoid cells in which he found purulent secretion and from that time the man was relieved of all complaint.

Thus we see the Roentgen photography may be of great support in the diagnosis of sinus disease and yet there are cases which are not discovered even with Roentgenography. The best proof of this is that the nine known cases of mucocele of the sphenoid sinus were not discovered before the sinus was opened.

In my case three rhinologists, among them the professor of rhinology in Leiden, declared that they could not find a single sign of sinus disease. The Roentgenogram was, as you see, not clear. Gerber supposed that this is caused by the extreme thinness of the bones surrounding the dilated sinus. When the professor of rhinology opened the posterior cavities on my advice, a yellow green mucous fluid streamed out, and then he could with probe reach the roof of the sphenoid sinus and discover that the roof was already perforated so that the sinus was in communication with the cerebral cavity.

MacKenzie also agrees that even Roentgen ray examination is not always sufficient, for he writes:

All cases must also be submitted to X-ray examination by a radiologist experienced in the skiagraphy of the nasal sinuses. But even X-ray examination is not infallible, so that in the presence of serious orbitoocular trouble, such as optic neuritis, for example, the only satisfactory method of excluding suppuration of the sphenoid and posterior ethmoid cells, the sinus most likely to set up eye disturbances, is to operate and open them up.

So we must say, as second conclusion:

The rhinologist cannot say with absolute certainty that a person has no sinus affection.

RELATIONS BETWEEN OPTIC NERVE AND SINUS DISEASE.

How is it possible that the optic nerve is affected by a sinus disease? What pathway is followed by the active agents?

To get an idea of this, we are in want of histologic examination of sinus, optic nerve and surroundings in such cases and it is a great pity we possess only very few of them.

As a rule the patients do not die from their sinus disease, and when a patient with a sinus disease and optic nerve disease dies it is very seldom that an autopsy is permitted where the part of the skull which we require is to be removed.

So there is little chance that the scant quantity of histologic material will be increased.

This chance will be better for material from sinus tumors than sinus inflammation, though the inflammation is more frequent.

Fortunately we have at least three histologic examinations which are all very important: two of tumors of the sphenoid, one of a pansinusitis.

It is highly interesting to compare the first two; in both cases there could be observed in vivo an axial neuritis which caused central scotoma for several months.

Birch-Hirschfeld found in his case the typical degeneration of the maculopapillar fibers, while on the contrary De Kleyn and Gerlach found in their case, though the central scotoma had already existed for more than seven months, almost no change in the optic nerve. They found the veins and capillaries of the optic nerve and nerve sheaths more filled than usual and a little infiltration of some of the nerve sheaths.

These two cases are typical of the clinical processes of the optic nerve disease in sinus affection, for we know we must distinguish here two forms, one which improves directly after the opening of the sinus, the other which does not diminish or diminishes only to a point and remains there.

In the first form there may be, as in the case of De Kleyn and Gerlach, only small and reparable changes in the optic nerve, in the latter irreparable changes, as in Birch-Hirschfeld's case.

The clinical symptoms in the reparable cases may be caused by edema, stowage, toxins pressure and beginning inflammation, the irreparable by degeneration and atrophy as sequence of long continued inflammation or pressure.

De Kleyn and Gerlach's case is so highly important, because it shows that even after seven months of central scotoma nothing irreparable was found, so that we must never despair, be the duration never so long, but treat the cases as soon as we get a chance.

The other case of De Kleyn and Gerlach shows us how an inflammation can spread from the nose and nasal cavaties to the optic nerve and penetrate the bony separation wall. They found by histologic examination of a patient with pansinusitis ulcerations in the nose and lacrimal passage, infiltration around the epithelium of the mucosa of the sinus, and they saw, as I show you here, the infiltration penetrating into the narrow spaces of the bone and in other field the infiltration following a small vessel. Though by an unlucky accident a slide with sections was lost just at the field where the infiltration reached the optic nerve sheath, the other sections are so clear and distinct that we see how the inflammation takes its pathway from the sinus to optic nerve.

The optic nerve sheaths are infiltrated just at the spot where they are nearest to the sinus, and we see an interstitial neuritis optica start from the nerve sheaths.

The anatomic disposition may be of great influence whether an optic nerve will be diseased in the course of a sinus affection and we know, especially from Onodi, that the anatomic relations greatly vary.

Onodi showed us that the optic nerve may pass freely unprotected by any bony shield through an ethmoidal cell or even through the sinus sphenoidalis, while on the other hand in some cases the optic nerve is surrounded by a huge wall. This of course may be of great influence. If, for instance, a cell or sinus is filled with pathologic secretion the free optic nerve surrounded by this fluid may become diseased by it, and even if there is only slight or even no secretion at all, the nerve may still become diseased by direct contact, because it passes twice through the whole afflicted mucosa.

On the other hand, a swelling of the nerve may in sequence to toxic edema cause more disastrous effect if the nerve is enclosed in a bony canal; for the swelling may even give rise to such a pressure that choked disc or atrophy may result.

Moreover we know that the bony separation wall is not an absolute protection against invasion of inflammation, for we saw in the case of De Kleyn and Gerlach where pansinusitis gave rise to an optic nerve disease, that the inflammation can penetrate the wall and cause an inflammation of the optic nerve sheaths and of the nerve itself. So we cannot say that the protection of the nerve by a bony wall is an advantage in every respect.

As a rule we take it for granted, that the chance for the nerve is the greater the nearer it is to the diseased sinus, so that we even think that a sinus which touches the optic nerve of both sides or of the opposite side only, may give rise to affection of both nerves or of the opposite nerve. How many possibilities there are with reference to optic nerve and sinus sphenoidalis is shown by the schema of Quix and the preparations of Onodi.

That indeed the anatomic vicinity is of great importance is proved by De Kleyn and Gerlach's case where we saw the affection of the optic nerve begin at the spot where it is nearest to the sinus.

Thus we discover that there exist different ways in which a diseased sinus may affect the optic nerve.

1. By direct spreading of the inflammation, as is proved by the De Kleyn and Gerlach's case.

2. By pressure caused the walls of the dilated sinus, as we see in the cases of mucocele where every nerve in the neighborhood may become atrophic from the pressure.

3. We suppose that bad influence may be exercised by toxins, edema, stasis, etc.

We must distinguish:

a. Reparable optic nerve affections, which may remain for months and months, caused by toxins, edema, stasis, slight inflammation, slight pressure, and

b. Irreparable optic nerve changes caused by degeneration and atrophy, in consequence of the same processes as mentioned above, but greater in degree.

To get a better view of the way in which the sinus disease involves the optic nerve we stand in great need, not only of more histologic material of sinus and optic nerve disease after the atrophy, but also of bacteriologic and histologic examinations of the material which the rhinologist gains by the operation upon the sinus, and it is to be hoped that every rhinologist will try to help close this unnecessary gap in our knowledge.

TREATMENT.

Here we must distinguish those cases of optic nerve affection in which there is a sinus disease affection and those which are of unknown origin, and where no sinus disease can be diagnosticated. The first cases are easy enough. Where we find a sinus affection in a patient with optic nerve disease we must treat the sinus. We know that a sinus disease may cause an optic nerve affection and therefore we have the right to suppose that if a sinus disease is present in a patient with an optic nerve affection of another origin, the latter may be aggravated by the former. Where this is the case we ought to treat every sinus affection in a patient with an optic nerve disease, even if this is not of nasal origin. I always should advise ophthalmologists and rhinologists to begin their treatment in a conservative way; many sinus diseases are cured with the cocain adrenalin spray.

The other category causes us on the other hand very much difficulty.

What to do when a patient has an optic nerve disease and the rhinologist does not find by careful examination the signs of a sinus disease, Roentgen rays included.

Of course the patient must be examined carefully for internal and neurologic diseases. When some other cause for optic nerve disease is found, I should still always urge, to consider the accessory sinuses. It is possible that a patient with multiple sclerosis, with diabetes, syphilis, tuberculosis, rheumatism or post trauma may also have a sinus disease. It would be very disagreeable to be contented with one cause for the optic nerve affection and overlook sinus disease which might be the real cause or an adjuvant. I remember a patient who was sent to me by an ophthalmologist, for optic nerve disease and purulent secretion in the nose, but the rhinologist refused to open the posterior sinuses because he did not find other signs of sinusitis and the patient had syphilis. According to the rhinologist the origin of the nerve disease was absolutely explained by the syphilis. They used antiluetic treatment alone until one eye of the unlucky patient was absolutely out, and the other almost blind. In Leiden, the rhinologist could find no other affections of nose and accessory sinus in the nose but when he opened the posterior sinuses of both sides, on my advice, there was purulent inflammation of both sinus sphenoidales and the posterior ethmoid. Soon after this the visual acuity of one eye recovered, becoming more than 1/2, but several scotomata remained and the other eye remained blind. A patient with multiple sclerosis, syphilis, tuberculosis, rheumatism or trauma may suffer from a sinus disease which has nothing to do with the affection, but he may have a sinus disease caused by the syphilis, the tuberculosis, the rheumatism or the traumatism; so that these diseases are not the direct cause of the optic nerve disease but the indirect, whereas the sinus affection is the direct cause. Therefore we ought always to examine the nose and accessory sinus in every patient with optic nerve affection, even if there is found another possible cause for this affection. When nothing at all is found, many authors say it will be multiple sclerosis because this disease is thought to be in 50 to 70 per cent the real cause of retrobulbar neuritis and the optic nerve affection may precede every other symptom 10 or more years. Even if this is right we cannot let our treatment be influenced by this consideration.

When there is an optic nerve disease without any other symptom of multiple sclerosis, for us the patient has no multiple sclerosis, at least we cannot perceive it, and we must treat him as if he had no multiple sclerosis until he gets other symptoms of this disease.

What to do in those cases of optic nerve disease without known origin is very difficult to determine, and a very close cooperation of rhinologist and ophthalmologist is required. We see in other countries how necessary people think it to discuss this problem in joint assemblies of rhinologists and ophthalmologists, as was done in September of last year at Nauheim in Germany; in March, this year, in Vienna, Austria, and in May, in Paris, France. You Americans have the good luck to possess this academy, so that you can discuss this and other problems together as often as you like to do.

Now, there are ophthalmologists who say that when the ophthalmologist thinks it necessary to open the sinuses the rhinologist must obey; he is in this way the handcraftsman for the ophthalmologist.

I am not of that opinion. I for one think that an organ specialist is not only the man who knows most of his special organ, has the most practice in operating on it, but is also the man who must watch his organ as a precious treasure which is under his trust, so that he must guard it from unnecessary mutilation.

No surgeon of whatever kind has the right to operate, that means to mutilate an organ, when he is not thoroughly convinced that what he is doing gives the best chance to the patient; he must never operate on the responsibility of another doctor. This is as well the case in appendicitis, ulcus ventriculi, trepanation for tumor cerebri or in sinus disease. Of course the surgeon cannot master every special method of examination and so he must believe the internist when he tells the surgeon what he finds upon examination of the stomach and intestinal contents, the ophthalmologist when he tells him of the visual acuity, the visual field, the choked disc, the neuritis, etc., but when he examines the patient himself, and when both have communicated with each other what they found by their examination and what they think of the diagnosis then they must consult with each other about the possibility and the desirability of the operation in general and in every individual case. When the surgeon is convinced by the result of his own examination or by the arguments of the other physician, he has the right to operate, otherwise never.

We now return from these general remarks to the optic nerve and sinus diseases. We know that our treatment will be greatly influenced by the conclusions already made.

1. The ophthalmologist cannot say whether or not an optic nerve disease is of nasal origin.

2. The rhinologist cannot exclude with certainty the presence of a sinus disease; to those two we add:

3. Opening of the sinus, even if nothing pathologic is found in the sinus, may be of temporary or permanent good effect on the optic nerve disease.

I think this latter result may be produced by derivative treatment; it may be that the bleeding or the change of the lymph stream is the cause, so that it acts as a bleeding or as installation of dionin or subconjunctival injections.

When we ask the ophthalmologist must the sinus be opened and he views the question only from his narrow point of view as organ specialist, he must say yes, as the eye has nothing to fear from such an operation and much to gain, whether or not a sinus disease is discovered.

If, on the other hand, the rhinologist is asked, must we operate? He must say as organ specialist no, as the nose is mutilated by the operation and there is only a little chance that the nose has to gain anything, that is, in the case that a sinus affection is discovered. Then they must consult together, but the ophthalmologist should not say, as v. Eicken describes, this patient will soon get blind, if you do not open the sinus and his eyes will be saved if you do. Then the rhinologist has no choice, but he is persuaded by unfair means, as no ophthalmologist can say whether a patient will get blind or will stay blind, or if he will recover after the operation.

Our treatment will of course be influenced by the eye symptoms; if a patient is blind or nearly blind no rhinologist will refuse to give him a last chance, but the greatest difficulty is in the chronic cases.

Here the rhinologist must ask the ophthalmologist what can be gained.

What do you know from literature as to the frequency and the amount of recovery of the visual power after the operation and of the number of cases in which a sinus disease is found and what is your personal experience in this? They know what is gained usually and what is the personal experience of the ophthalmologist. On the other hand the ophthalmologist must ask the rhinologist what is the damage which can be done by the operation. I know that it is possible in an imprudent operation to curette away an optic nerve which passes freely through a sinus; this can be prevented. In what per cent is damage done after the sinus operation, and what kind of damage, and what is your personal experience? The per cent figures gathered from the whole literature may decide whether in general the operation is tolerated. Say for instance that in every 10 sinus operations in optic nerve disease of unknown origin we find once a sinus disease and once the visual power is considerably increased, then we must ask ourselves whether we have the right to operate unnecessarily on eight patients to help two. The personal experience of both specialists is of influence too, as the experience of the ophthalmologist shows whether he has a good clinical view in choosing the patients upon whom an operation is to be performed, the experience of the rhinologist showing whether he has good luck as operator or not.

If they both know the possible advantage and possible damage, they have to decide whether or not in a special case they will operate and in this regard the personality of the patient will be of value, as we must always keep in mind that a sinus operation is not an indifferent matter for the patient.

Though we can make the nose tolerably anesthetic, every patient thinks a sinus operation is very disagreeable and for a nervous patient it is a terrible shock, so that we cannot deny the influence on the psyche, especially when the operation has no effect. In strong minded people we may discuss as far as possible the question with the patient himself, in others with the family. Thus one of them should know that there is no question of an operation for an existing disease but an exploratory operation for a possible disease, otherwise they will always think if no good result is gained that the operator failed to do his duty.

Acting in this way I have never met a rhinologist who refused a sinus operation when I wished him to do it and I may say that I was very lucky in the results, so lucky that I hope I will always have the same experience.

You see from these considerations what is failing; we want urgently, statistics of the results of the operation as well as to the direct and late results for the visual power and of the results of the damage to the nose, but also statistics of what happens with the eyes of the patients in which the sinus is not opened. To make these statistics it is necessary that we all use the same terms, which I am not convinced has applied. We see that in Nauheim Von Eicken compares statistics of Heine in Kiel, who found in 50,000 patients 46 cases of retrobulbar neuritis, that means in 0.092 per cent, and among them only 3 cases of nasal origin, that means 0.006 per cent, and the statistics of V. Grosz in Budapest, who found in 18,587 patients 58 cases of optic nerve disease of nasal origin, that means in 0.312 per cent or 52 times as frequent. V. Eicken suggests that this considerable difference may be caused by the fact that V. Grosz perhaps reckons to be of nasal origin every case in which the visual power is increased by an endonasal operation. But if this was the case it must still be explained how it is possible that V. Grosz has 3.5 times more optic nerve disease of nasal origin than Heine in retrobulbar neuritis of all origins. Either the optic nerve disease must be much more frequent in Budapest than in Kiel, or V. Grosz and Heine do not use the same terms.

So I think the first thing necessary is to get uniformity in the nomenclature and then try to get statistics of the advantages which the eye gets from the sinus operation and of the damage which the nose gets from this operation.

I hope, Mr. Chairman, ladies and gentlemen, that you will all help to develop such statistics in order to solve the highly important question, as to whether we must operate upon the sinuses in optic nerve disease of unknown origin.

XXI.

RECENT OBSERVATIONS ON LARYNGOPULMONARY TUBERCULOSIS.*

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LIBERTY, NEW YORK.

We all realize how difficult it is to render an accurate prognosis in laryngopulmonary tuberculosis. The average length of life of a patient suffering from laryngopulmonary tuberculosis has been estimated by various men to be from two to four years. Although this may be a fact, to the individual patient it means very little, for we all know that some cases of laryngopulmonary tuberculosis live but a few weeks, while others enjoy good health for many years. The individual affected is not particularly interested in the average length of life, but he does want to know how long he may expect to live. In order to arrive at a fairly accurate prognosis it behooves us to study each case by itself, and even then we may expect to err occasionally.

It is generally understood that laryngeal involvement renders the prognosis very serious. Older writers have been of the opinion that laryngeal involvement rendered the prognosis absolutely hopeless. My personal observations are at great variance from the generally accepted opinion, and it is my present purpose to bring forth some of my observations based on nearly ten years' experience with tuberculosis of the lungs and of the larynx.

While on the staff of the New York City Sanatorium at Otisville, New York, from 1913 to 1918, I observed a large number of patients with laryngeal tuberculosis, some of whom lived only a short time, while others lived and enjoyed good health for years in spite of the fact that their lesions were quite extensive. This puzzled me very much, and therefore I examined a large group of patients, carefully noticing the pathologic manifestations. I also observed these patients for a long period, making careful record of the course of the disease in the larynx. From these observations I became convinced that laryngeal tuberculosis was either peracute, acute, subacute, or chronic. These various forms have already been discussed by me in previous works, but because of the intimate bearing on the subject I will describe them again.

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*Read at the Twenty-sixth Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology, October 17, 1921, Philadelphia, Pa.

The peracute cases of laryngeal tuberculosis, fortunately, are infrequent compared with any of the other forms. These cases are very rarely seen in the stage of edema, but usually present extensive ulceration and perichondritis. They are often accompanied by tuberculosis of the faucial tonsils and soft palate. Dysphagia and aphonia are always present, especially when the pharynx is involved. Emaciation is rapid, the patient is febrile and soon becomes moribund. Other organs of the body, such as the intestines and meninges, are often also involved. The peracute type usually begins as such, although occasionally it follows the acute type.

The acute type is characterized by a soft edema of the larynx with a tendency to ulceration. It may begin as such, or it may follow the subacute or chronic type. It usually occurs in the advanced cases of pulmonary tuberculosis, often in moderately advanced cases, and occasionally in incipient cases. There is little or no tendency to fibrosis. Nodules consisting of tubercules can often be seen projecting above the surface of the mucosa. When the tubercles caseate they give rise to ulcers. During the stage of caseation the nodules often coalesce, with the production of extensive ulceration. The patient usually suffers from marked hoarseness or aphonia, a sensation of fullness in the throat, and irritation with a constant desire to expectorate. Dysphagia is of common occurrence and may be very pronounced, depending on the location and character of the ulcerations.

The subacute type is characterized by a semiedema of parts of the larynx with a moderate tendency to fibrosis. It may begin as such or else follow the acute or chronic type. It takes a subacute course, has a tendency to healing and is usually benefited by treatment. Lesions with papilliform infiltrates and soft polypoid excrescences generally belong to this type. The local symptoms may be slight or moderate, depending largely on the site and the extent of the lesion. Hoarseness, as a rule, is present, particularly when the true vocal cords or the interarytenoid sulcus is involved. Dryness of the larynx is generally complained of, and more so when the fibrosis is extensive.

The chronic type offers the best outlook, and is characterized by firm infiltration of part or parts, with a marked tendency to fibrosis and healing. The lesion, due to an extensive proliferation of connective tissue cells, is usually limited in extent to the affected areas, and may remain so for an indefinite period. These patients suffer very little, and often, with the exception of occasional dryness in the larynx and slight hoarseness, have no symptoms at all. For this reason this type is very often overlooked.

In rendering a prognosis in laryngopulmonary tuberculosis, although all other factors, such as character and extent of the pulmonary lesion, the general condition and mental condition, early diagnosis, etc., should be considered, the chief determining factor is the character of the laryngeal lesion itself. For it very often happens that a patient will be given a good prognosis based on his pulmonary lesion, only to have the prognosis changed when it is discovered that he suffers from an acute involvement of the larynx as well.

The summary of my observations on the prognosis in laryngopulmonary tuberculosis, therefore, is as follows:

In peracute cases, with or without involvement of the pharynx and regardless of the pulmonary lesion, the prognosis is bad, and the life of the patient is from a few weeks to a few months. Here, as a rule, the patient dies from an overwhelming toxemia and from inanition caused by dysphagia. Local treatment is of no avail and is not indicated except for the relief of pain. Our aim should be to make the patient as comfortable as possible.

Acute cases often improve under skillful treatment, and when the pharynx does not show macroscopic involvement and the pulmonary and general conditions are at all favorable, the prognosis is not hopeless. These patients should be kept under a careful regimen and persistently treated generally and locally. They often improve and occasionally become completely cured.

The subacute cases present a good outlook, and the prognosis depends a great deal on the pulmonary lesion and timely treatment. With good general care and with proper local treatment the laryngeal lesion becomes fibrosed and chronic.

The chronic cases present the best outlook. Patients with this type of lesion usually get along best under general treatment alone. The prognosis as to life is hardly ever influenced by chronic laryngeal tuberculosis, and therefore in rendering a prognosis in these cases the larynx may be entirely disregarded.

I shall now illustrate my paper by demonstrating to you a few plates of the different types of laryngeal tuberculosis:

Case Record No. 1.—Mrs. R. G., age 20. Patient consulted me on account of slight hoarseness and dysphagia. Examination revealed early pulmonary tuberculosis. somewhat active. Larynx showed edema of epiglottis and both vocal cords, and a semiedema of both arytenoids. Patient also gave a history of a six months' pregnancy. General and local treatment were instituted but apparently without result. Dysphagia became worse and emaciation progressed. About two weeks later both tonsils revealed the presence of macroscopic tubercles and gradually the soft palate and uvula became involved. The disease progressed rapidly and patient died about twelve weeks from the time I first saw her, giving premature birth two days before death.

Diagnosis: Peracute tuberculous laryngitis with involvement of tonsils and soft palate. Fig. 1.

Case Record No. 2.—R. M., aged 36. Pulmonary lesion advanced and active with bilateral involvement. Larynx showed very acute edema of epiglottis, arytenoids and aryepiglottidean folds. Patient has a great deal of dysphagia and suffers from complete aphonia. Local treatment has been attempted but with no results. Diagnosis: Peracute tuberculous laryngitis; prognosis, very bad. Fig. 2.

Case Record No. 3.—E. N., age 23. Patient was referred to me by Doctor Thomas R. French, of Brooklyn, New York, in February, 1921. First examination revealed that he suffered from advanced pulmonary tuberculosis with activity. He ran an afternoon fever of 101-102; was considerably emaciated and suffered from loss of appetite. Examination of larynx showed acute edema of epiglottis with angry looking ulceration of right side. Rest of larynx seemed quite free. Dysphagia was pronounced. Patient was put to bed where he was kept for about four months and larynx was treated locally. Examination in early part of September showed that pulmonary lesion was quiescent; temperature was normal; patient gained about five kilograms in weight; ulcer of epiglottis was almost completely healed, with some loss of tissue and formation of scar; dysphagia completely relieved and patient allowed to return to work. Diagnosis: Acute tuberculosis of larynx, healing by fibrosis. Fig. 3.

Case Record No. 4.—M. H., age 30. Referred to me by Doctor V. G. Bourke, of Livingston Manor, New York. First examined February 2d, 1921, and her condition was as follows: Pulmonary lesion—Far advanced and moderately active; sputum positive. Larynx revealed inflammation and uniform infiltration of epiglottis with ulceration; infiltration with erosion of both aryepiglottidean folds; edema in the posterior commissure and pear shaped swelling with ulceration of both arytenoids. There was also an acute infiltration of posterior ends of both vocal cords. Patient ran a fever in the afternoon and suffered from great dysphagia and aphonia. The laryngeal lesion was apparently of the acute type. General treatment was instituted, and in addition to vocal rest the patient was treated with topical applications of solution iodin. On October 5, 1921, patient showed general improvement; pulmonary lesion was considerably more quiescent; examination of the larynx showed the epiglottis only very slightly edematous, and on a casual examination it might even be considered normal; the arytenoids showed only a slight thickening, with healing ulceration. All the edema observed eight months ago was practically gone. No dysphagia, and patient now has a pleasant low pitched voice. Diagnosis: Acute tuberculosis of the larynx, gradually healing by fibrosis. Fig. 4.

Case Record No. 5.—J. F., age 24, is a case of far advanced pulmonary tuberculosis with positive sputum; marked hoarseness. Examination revealed extensive infiltration of both cords with large papillomatous mass in posterior commissure, interfering with complete approximation of cords. Diagnosis was subacute tuberculosis of the larynx. Patient has been treated locally with topical applications of solution iodin in increasing strengths and is showing steady improvement in voice. Fig. 5.

Case Record No. 6.—L. R. Patient has a moderately involved pulmonary lesion with positive sputum. Examination of larynx shows a papillomatous growth in posterior commissure which has been stationary for over a year. He is practically free from symptoms, except for occasional discomfort and feeling of irritation in larynx. Such a lesion practically never influences the prognosis. Diagnosis: Chronic tuberculous laryngitis, with a good prognosis. Fig. 6.

Case Record No. 7.—M. F., aged 19. Referred to me by Doctor Thomas R. French, of Brooklyn, N. Y. First examined on March 12th, 1921. Findings were as follows: Pulmonary lesion incipient and inactive; sputum positive; examination of larynx showed a warty growth of epiglottis and a general hyperplasia of the mucous membrane of the entire larynx, with thickening of the arytenoid cartilages; a small portion of the rim of the epiglottis was recently excised for diagnosis, leaving a large slowly granulating ulcer. Patient suffered from much dysphagia. The patient, in addition to general treatment, was also treated by local applications of solution iodin, and on September 2d the examination showed the epiglottis much diminished in size and edema generally lessened. The entire larynx showed distinct improvement with complete relief from dysphagia. Diagnosis: Lupus of larynx, gradually healing. Fig. 7.

Case Record No. 8.—J. C., age 38. Admitted to the Otisville Sanatorium in 1913 and was found to have a moderately advanced pulmonary lesion with a positive sputum. Wasserman negative. Larynx showed a tuberculoma of interarytenoid space with healed erosion, thickening and infiltration of both cords, especially the left. Left cord also showed healed ulceration on its upper surface, infiltration of both ventricular bands, especially the right, which almost entirely covered the right cord. Lesion showed complete fibrosis, and outside of marked hoarseness patient had no discomfort. The diagnosis was chronic tuberculosis of the larynx.

Examination of the larynx three and one-half years later showed the findings to be identically the same. In the meantime the patient has been doing very well and shows no signs or symptoms of decline. The probable duration of the lesion according to the patient's history is about ten years. The patient does not receive any local treatment.

I have not seen this patient in the past four years, but in answer to an inquiry from me, received last week, he states that he works, has gained some weight, and feels as well as ever. His voice is still husky, but with the exception of occasional irritation in the throat he does not experience any troublesome symptoms. Fig. 8.

Case Record No. 9.—I. T., age 20. Admitted to the Otisville Sanatorium on December 26, 1916. Chest examination revealed a moderately advanced lesion of the lungs. On the third day at the sanatorium, the patient developed a slight afternoon fever, which afterwards became persistent, rising to 101-102° F. on every afternoon, while the pulse ranged between 110 and 130 during his entire stay at the sanatorium. The examination of the larynx, on admission, showed a uniform infiltration of the epiglottis, with slight thickening at the posterior commissure and arytenoids. On January 5th the patient complained of slight dysphagia, and the examination showed the beginning of tuberculous infiltration of the oropharynx which kept getting more extensive, while the symptoms were gradually becoming more troublesome. On February 1st the patient complained of severe dysphagia and regurgitation of liquid food through the nostrils. He then showed extensive infiltration and ulceration of the oropharynx and fauces. Uvula was edematous. Larynx showed edema of epiglottis and both arytenoids, and the Wasserman test was negative.

On February 14, 1917, the lesion was widespread. Uvula was much swollen and covered with macroscopic tubercles. The lingual tonsils were also ulcerated. The larynx showed acute edema with macroscopic tubercles of all parts. All during his stay at the sanatorium the patient received local applications of solution of iodin to the affected parts, with no apparent benefit. On February 15th the patient was transferred to a city hospital, where he died a few days later.

This is a typical case of the peracute type and the prognosis is extremely grave. Fig. 9.

Case Record No. 10.—C. B., age 20. Pulmonary lesion moderately advanced with positive sputum. Laryngoscopic examination showed infiltration with superficial ulceration of the epiglottis; also infiltration with ulceration of both true cords ventricular bands and arytenoids. There was also marked interarytenoid hyperplasia with ulceration. Patient suffered from extreme hoarseness and sense of fullness in the larynx but there was no dysphagia.

The second picture shows the same patient after seven months of treatment. The edema had almost entirely disappeared; ulcerations, with the exception of those in the posterior commissure, are all healed. There was marked improvement in phonation, and the lesion showed steady healing by fibrosis.

The same patient was seen by me recently, about seven years after my first examination. The appearance of larynx is about the same as in the second picture; the epiglottis is absolutely free from involvement; the patient is still somewhat hoarse, but generally seems to be in fairly good condition Diagnosis: Acute tuberculosis of the larynx which is gradually healing by fibrosis. Fig. 10.

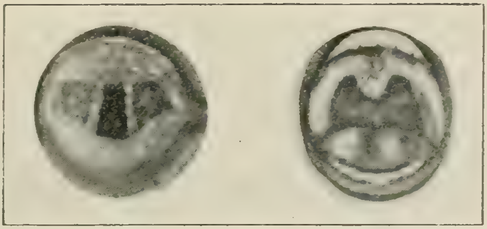


Fig. 1.

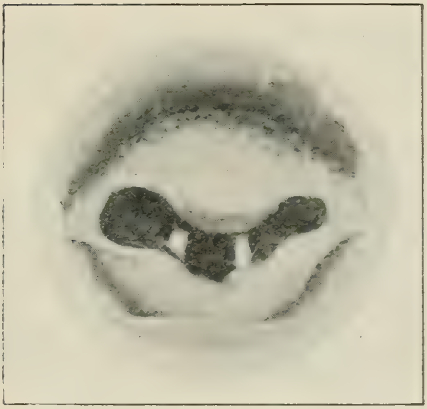


Fig. 2.

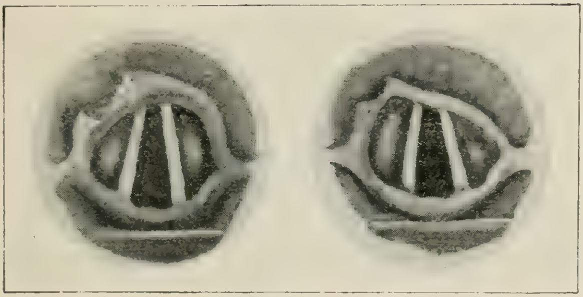
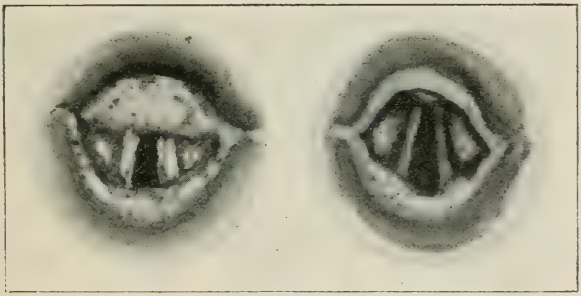


Fig. 3.



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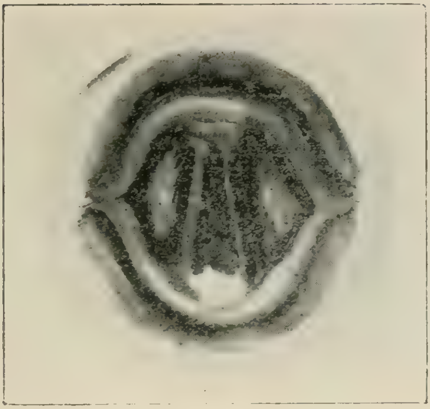

Fig. 5.

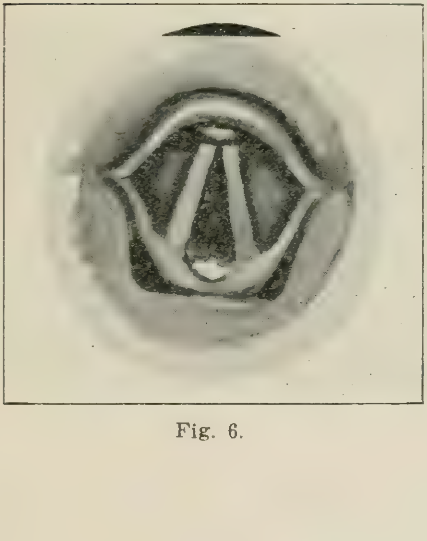

Fig. 6.

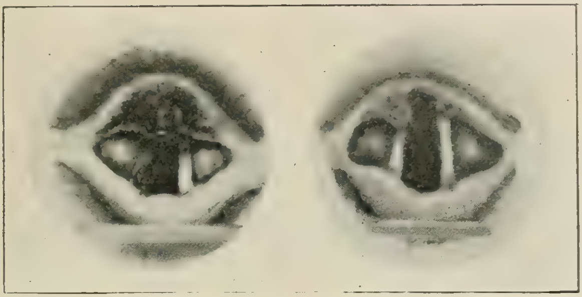

Fig. 7.

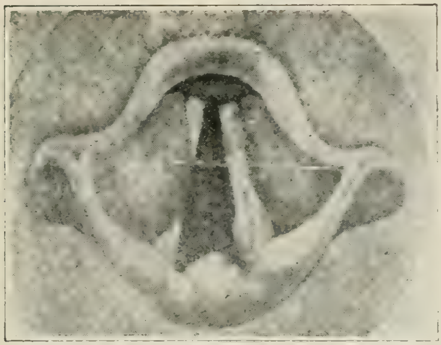

Fig. 8.

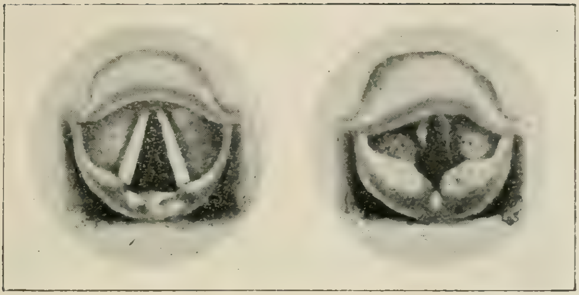

Fig. 9.

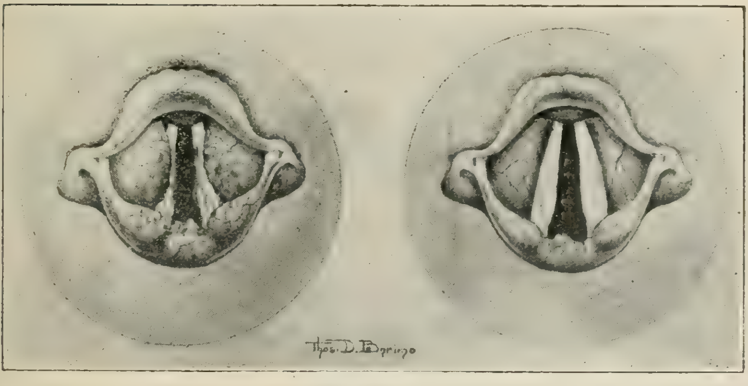

Fig. 10.

XXII.

THE USE OF EAR PLUGS IN AVIATION.

By Major Lloyd E. Tefft, Medical Corps, U. S. A., and
Elizabeth K. Stark,

DEPARTMENT OF OTOLOGY, MEDICAL RESEARCH LABORATORY
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Mitchel Field, L. I., N. Y.

Everyone who has flown for a considerable period of time, especially in bimotored ships, is familiar with the bothersome after-ringing in the ears and partial deafness lasting for several hours. These disturbances are caused by the sharp crack of the exhaust from the airplane motor, and if this condition is endured there will develop, eventually, more or less permanent impairment of hearing, inasmuch as the inner ear is a delicate organ and will not tolerate abuse without showing effects. The temporary effects of this exposure of the ear to massive sound are the ringing and temporary deafness, as stated above. In addition, however, if the abuse is continued, the temporary deafness may result in permanent partial deafness. It would seem wise, therefore, to remedy this condition, if possible, in order that the air life of the flier may not be shortened.

Three possible means of accomplishing this present themselves. One is the abolishment of the exhaust noise of the motor by a muffler, inasmuch as this sharp crack of the exhaust is the sound that causes the trouble. The remaining noise of the motor, the noise of the propeller, struts, etc., is either too low pitched or of not sufficient intensity to be harmful. Airplane motors have been successfully muffled, but at the expense of some of their efficiency, so that fighting planes will probably remain unmuffled. Secondly, a cabinet, nearly soundproof, can be constructed which will eliminate enough of the noise. This has also been done, but again at the expense of the efficiency of the airplane. It would seem, therefore, that at the present time nothing can be done with the airplane itself to eliminate enough of the motor noise. This being true, the fliers themselves offer the only means of abolishing this menace. For this reason, various substances were placed in pilots' ears to determine if one could be found which would not interfere with the flier hearing his motor, which would not hurt or annoy the pilot in any way, which could be easily and readily inserted or removed, which was reasonably inexpensive, and which would eliminate enough of the sound so that the flier would have no bothersome after-ringing or deafness.

Most fliers use cotton in their ears. This helps some, but does not usually abolish sufficient sound, and becomes easily displaced. Rubber plugs of various sizes and shapes satisfy certain fliers, but, due to the difference in the shape of the ear canal, a plug that will fit one man's ear will not necessarily fit the ear of another, hence, if used, will result in pain, discomfort or displacement if too large, and in insufficient protection and displacement if too small. In fact, any solid substance which cannot be moulded to the outer ear canal, even if composed of material as soft as rubber, must, in order to be efficient, be placed in so tightly that it will cause irritation of the delicate membrane lining the canal. Consequently, research work was undertaken here to find some substance, pliable enough when warm, to mould itself into the form of the outer ear canal and hard enough to retain its shape after being so moulded. Various substances, like parresine, beeswax, printer's wax, and gum were tried. The Navy Aviation Medical Service had previously tried parresine alone with some success. We found here that better results were obtained with a mixture of parresine and beeswax: five parts of parresine with one part of beeswax for summer use, and four parts of parresine with one part of beeswax for winter use, as beeswax softens the mixture and more of it is necessary in colder weather. Ear plugs of this mixture seemed of the right consistency, but lost their form too readily; hence, solid substances were used as a core to give "body" to the plugs, make them hold their shape better, and permit of easy withdrawal. Absorbent cotton does not possess the necessary resiliency and becomes too easily packed. After considerable experimentation it was discovered that lamb's wool (surgical wool) was ideal. It does not pack as easily as cotton, yet is readily moulded; consequently the plugs hold their shape better and can be easily withdrawn.

In order that a large number of plugs can be made at one time, moulds were prepared from plaster of Paris blocks. These moulds are in two sections, thus enabling the completed plugs to be easily removed. Figure 1 shows a photograph of one of these sections, and from it some idea of the shape of the plugs can be ascertained, as well as from Figure 2, a photograph of an actual plug.

In making the plugs we first grease the moulds with a thin coating of vaseline; this prevents the wax from adhering to the mould. The two sections were then apposed and a small tuft of lamb's wool is placed in each depression. The melted mixture of parresine and beeswax, in the proportions required, is next poured in and allowed to cool, after which the moulds are separated, the plugs removed and any irregularities in their shape smoothed off. For shipping, the plugs are packed in an ordinary pasteboard box.

The plugs must be warmed and moulded before inserting. This is accomplished by holding a plug in the palm of one hand and kneading it with the fingers of the other hand. When it is soft enough, it is moulded into about the form depicted in Figure 3, and is then ready for insertion. When the plug is placed in the ear, the main thing to be observed is to avoid having the conical end so long that it will impinge upon the ear drum. This will not do any material damage but will cause discomfort when inserted. The plug is firmly inserted with the conical end in the external ear canal, and the remaining flat portion moulded over the end of the canal in the ear outside. A pledget of absorbent cotton, or, preferably, wool, about the size of a small egg is next placed in the ear on top of the plug and the helmet drawn down over this. This cotton or wool serves the purpose of maintaining constant slight pressure upon the plug, thus preventing its becoming dislodged by vibration. Withdrawal of the plug is easily accomplished by engaging any portion of it with the finger nail. Figure 4, the photograph of a withdrawn plug, shows the general shape which the plugs assume in the ear.

Several hundred of these plugs were sent to each of the flying fields throughout the country, and the Flight Surgeons of these fields were requested to report upon their use, adding any suggestions or criticisms that they or others cared to offer which would make for a better plug. A number of letters have been received from these Flight Surgeons stating that while the plugs are not in universal use at their fields, those fliers who had used them were very much pleased with them. A number of pilots still share the belief that any artificial means tending to preserve their health must not be used because they might be adjudged to be "soft" or timorous. This attitude, however, is becoming less and less prevalent, and it is believed that in time ear plugs of a type similar to those described above will constitute part of the equipment of every flier.

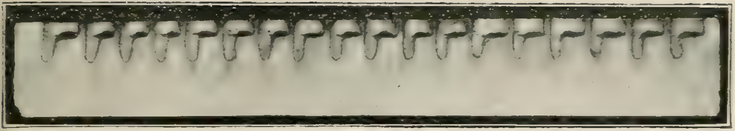

Fig 2. Fig. 3. Fig. 4

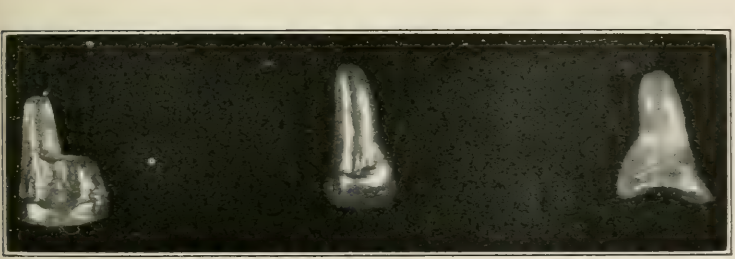

Fig. 1.

XXIII.

LARVAE OF THE FLY SARCOPHAGA (SARCOPHILA) WOHLFAHRTII IN THE EAR.

By Alexander Zebrowski, M. D.,

New York.

It is well known that the number of foreign bodies, both animate and inanimate, met within the external ear channel is very great. In every textbook of otology we find enumeration of all possible foreign bodies found within the external ear. Kerrison[1] in his book says "there is hardly a limit to the variety of foreign substances which may be introduced, either intentionally or by accident, into the external auditory canal." In the chapter on the foreign bodies in the ear the authors all emphasize the importance of skill and care in the removal of foreign bodies, stating that no foreign body can do so much harm as a careless and unskilled trial to get the foreign body at any price. Except in the textbook of Ballenger[2] I have nowhere found a notice that an animate foreign body—e. g., the larvæ of insects—can occasionally endanger the life of its victim, although in the majority of textbooks on otology we find mention of larvæ in the human ear.

As a curiosity I cite a statement of such an authority as Bezold,[3] who in his textbook denies the possibility of the presence of larvæ in the human ear.

Nevertheless, it is a fact that at least one kind of fly, namely Sarcophila or Sarcophaga, described at first in the eighteenth century by a German physician, Wohlfahrt,[4] can cause most severe pain and under certain circumstances may endanger life itself. Wohlfahrt had a patient 67 years of age, who for eight days suffered horrible pains in the head. The regions of the right eye and nose were swollen. There was epistaxis from both nares which lasted several days without cessation. One larva fell from one nostril, and when the patient, according to the advice of Wohlfahrt, poured some alcohol into the nose, 18 larvæ came out and all symptoms disappeared very soon.

Wohlfahrt examined the larvæ, and after some weeks of waiting there appeared a cocoon and a fly, which he has described and very instructively illustrated (Fig. 1). This observation for a long time remained unknown and the German entomologist Schiner described the same fly in 1869 under the name Sarcophila magnifica, without mentioning the description of Wohlfahrt. A prominent Russian entomologist, Portchinsky,[5] claimed that this fly should bear the name of the discoverer and proposes to give her the name "Sarcophila Wohlfahrtii," or "Wohlfahrtia magnifica." In his very interesting monograph he gives all the data from the literature, describes the fly, its customs, and cites a great number of cases where the larvæ have penetrated into the human body, for the most part, however, into the ears, nose and eyes.

The fly is very common in Southeast Russia, in Southern Siberia and Turkestan. Its habitat is near herds of cattle, chiefly sheep, on which it deposits its larvæ (not eggs, but larvæ), for the most part in even the smallest wounds. The cattle owners in that part of Russia know very well the dangerous "worms" on cattle, from which the animals sometimes die, but, according to Portchinsky, they did not know that the worms are descendants of a beautiful big fly, grayish in color, which Portchinsky has repeatedly found in the neighborhood of the cattle. The fly appears only on hot sunny days; at other times it is hidden The female flies have a very delicate sense of smell, and even the smallest wound attracts them. No wonder that when a child with a purulent discharge from the ear is sleeping in the open the fly, when present, will come very quickly and put her larvæ into the ear.

The larvæ when born are small, but very active, and grow rapidly. They have on the head end an apparatus consisting of three strong sharp hooks (Fig. 2), with which they penetrate into the tissues of the body and make deep, long channels, causing inflammation with profuse discharge. This discharge serves as food for the larvæ. In the second stage of the development they lose the middle hook, become quite large (up to 15 mm. length) and then leave the animal. Then they burrow in the ground, form a "false" cocoon and in two weeks a fly is born.

After this introduction I shall turn to the description of the cases observed and treated by me in the years 1915-16, when I was practicing in the Crimea. My first case came to me in September, 1916 (this month in the Crimea is very hot). During my office hours a whole Tartar family came to me, bringing a boy 10 years of age who was crying, yelling, groaning and clutching at his head. Sometimes he was even swooning. The parents told me that for four days the boy had not slept a wink, complaining of terrible pains in the ear, which had been discharging pus for three years (after measles). They had seen what they described as large worms in the ear. They removed some of them, but some are still there, and the country physician could not get them out by syringing with water.

The aspect of the left ear was awful. The whole auricle was reddened, swollen and very painful to the touch. In the meatus several larvæ were to be seen. When I tried to pick some of them with the ear forceps they burrowed quickly into the depth of the meatus and other larvæ came into view. The tragus and lobulus were covered with pus, blood and black spots. The mastoid process was tender on pressure, especially in the fossa mastoidea. The lymphatic glands on the neck was enlarged and swollen. Temperature, 102°. The pulse, weak, 130. The boy appeared to be very ill.

At first I tried to syringe the ear with water, but only one larva came out. Seeing such a thing for the first time in my life, I examined it with the magnifying glass and discovered the hooks mentioned above. The larva made quick movements with the head and whole body which did not cease even when I put the larva into H_2O_2 and alcohol. Knowing that olive oil makes the removal from the ear of living insects (bugs, cockroaches) easier, I filled the ear with oil, which I always have on my office table for softening hard cerumenous plugs. Some seconds after that the larvæ appeared, one after the other. The oil had closed their breathing organs, forcing them to escape from the ear channel. Altogether, eleven larvæ, about 10 to 12 mm. long, came out. The child at once became quiet and fell asleep. After careful cleansing I examined the ear and found very remarkable changes. The skin layer of the auricle and external ear channel was red and swollen, with small excoriations. The remnants of the drum membrane were irregular in shape and covered with blood. In the large perforation I found one larva, which I removed with the ear forceps. This larva moved sluggishly. The malleus was in situ. The Shrapnell membrane was red, covered with blood, but no perforation. A strip of gauze moistened in the solution of liquor aluminum acetate was introduced and bandage applied. Next day the boy was very gay, stating that his ear was now quite well, but that he still has some pain in the ear at times. Temperature normal. No pain over the mastoid. After a week the perforation became smaller and two weeks later was replaced by a scar.

Obviously we had to deal here with absorption of pus from the drum cavity and antrum, caused by the presence of larvæ in the external ear. The larvæ were carried by a sarcophaga fly directly into the ear when the boy slept in the open on a sunny hot day. The chronic otitis after measles has, as we know, a tendency to be cured when proper treatment is applied. I think that the irritation of the edges of the perforated drum membrane caused by scarification accelerated the healing process.

The larvæ I sent for examination to Prof. Z. Mokshetsky (Mokrzecki), a Polish entomologist, the founder and director of the Crimean Museum of Natural History in Simferopol. He recognized them at once and was very pleased to find two larvæ still alive, from which he got cocoons and flies.

The same month I observed the second case, a Tartar girl. three years of age, with larvæ in both ears. The child slept in the open. Purulent discharge from both ears. I removed altogether 13 larvæ, with this difference, that I syringed the external ear channels with 2 per cent watery solution of chloroform. Chloroform, even in so weak a solution, kills the larvae very quickly, and is quite comfortable for the little patient. When applied per se it causes pain by burning the skin. In this case both drum membranes and ossicles were destroyed. After the removal of larvæ the child, who was yelling, crying, etc., as in the first case, became quiet at once. I saw her no more.

Next year (1917) I had three similar cases. All of them were children, and in all the chronic otorrhea was present. After removal of larvæ all these three cases recovered, although in one case the rise of temperature and weak, accelerated pulse, lasted for several days. In no case was an operation on the mastoid indicated, although in all I noticed a tenderness in the region of fossa mastoidea and a rise of temperature.

Among the cases cited by Portchinsky, we find two which ended fatally. In one case (larvæ in the ear, l. c. page 51) a boy 13 years of age, who had had otorrhea for a long time, slept outdoors and was infected by sarcophaga. Four days later, after terrible pains in the ear and in the head, the ear was syringed with milk and turpentine oil, and 30 larvæ came out. Nevertheless, the general condition of the boy became worse, and on the seventh day the boy died with symptoms of meningitis.

The second fatal case was that of a girl, 11 years of age, who suffered from ozena. After sleeping outdoors the child noticed discomfort in the nose, afterwards itching and pain, which grew worse every hour. Some days later a large quantity of stinking fluid escaped from the nose. After eight days of intense suffering the child died from meningitis. The postmortem examination revealed large destruction in nasal cavities, which communicated with orbits. The eye muscles were destroyed. In the nasal cavities altogether 58 larvæ were found.

When we bear in mind that the sarcophaga fly produces, according to Portchinsky, 124 to 168 larvæ, which are very hardy and active, there should be no wonder that under favorable circumstances they may endanger the ilfe of the victim. When, for instance, a dehiscence in the roof of the tympanum and antrum is present it is quite possible that in the absence of the drum membrane the larvæ can penetrate through the dura into the cranial cavity, causing meningitis directly. On the other hand, the larvæ may evoke the absorption of pus by blocking the external ear channel and thus cause an intracranial complication, if not removed at the right time. The best method for removal of larvæ is syringing of the ear with 2 per cent watery solution of chloroform.

REFERENCES.

1. P. Kerrison: Diseases of the Ear, 1911, p. 119.

2. Diseases of the Nose, Throat and Ear, p. 652. Ballenger writes about the Texas screw-worm fly (Compsomyia Lucilla macellaria), that this insect can be very dangerous to the life of his victim when the "worm" has penetrated through the bone into the cranial cavity, invading an ear affected with otorrhea.

3. Bezold: Lehrbuch der Ohrenheilkunde, 1906, p. 116. "In der Lehrbuechern finden sich auch durchgaengig Fliegenlarven erwaehnt, welche neben foetider Otorrhoe in knoechernen Gehoergang und der offenliegenden Paukenhoehle zur Entwickelung gekommen sein sollen. Da ein derartiges Vorkommnis weder von anderen neueren Autoren noch jemals von mir gesehen worden ist, so ist wohl ein Zweifel darueber erlaubt, ob hier nicht Taueschungen vorgelegen haben, welche durch raschen Wechsel von Lichtreflexen in der Tiefe entstanden sind."

4. Wohlfahrt: De vermibus per nares excretis Nova acta phys. Akad. Caes. Leop., 1770, p. 277.

5. Portchinsky, I. A.: Wohlfahrtia magnifica Schiner sive Sarcophaga resp. Sarcophila Wohlfahrtii Petersbourg, 1916 (in Russian language).

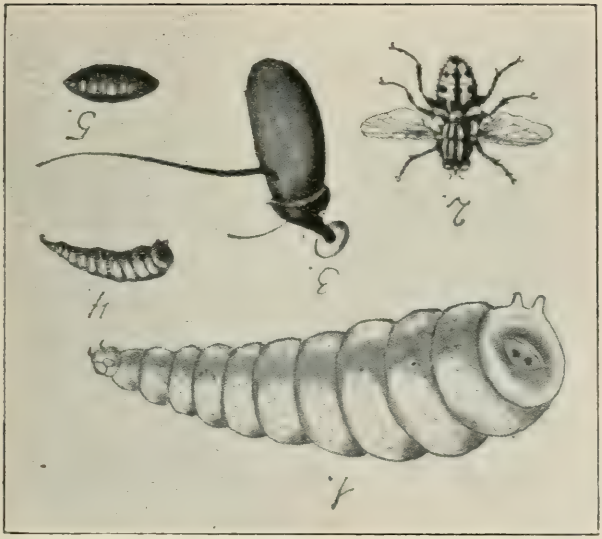

Fig. 1. The Fly of Wohlfahrt (Wohlfahrtia Magnifica).
1. The larva (magnified). 2. The fly. 3. Antenna. 4. The larva (natural size). 5. The false cocoon. (After Wohlfahrt.)

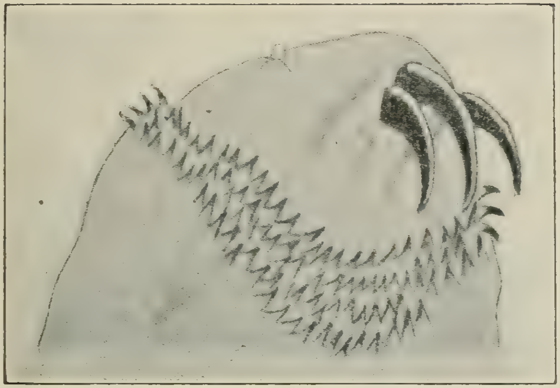

Fig. 2. The head end of the larva, magnified (after Portchinsky).

XXIV.

ADENOCARCINOMA OF THE MIDDLE TURBINATE, ANTRUM AND ETHMOIDS TWO YEARS AFTER RADICAL OPERATION WITHOUT RECURRENCE.

By David N. Husik, M. D.,

Philadelphia.

A. W., 32, white, machinist, married, was referred to the University Hospital dispensary on account of nasal obstruction and pain in the head and face.

H. P. I.: Patient began to complain of frontal headaches in 1915. These headaches would last about 24 hours and would recur almost every week. He gradually became accustomed to them. In January, 1919, he began to notice left sided nasal obstruction and intermittent pain in his cheek, which gradually became worse, and in May, 1919, presented himself for examination.

Nasal examination shows the left nostril completely filled by a large irregular mass, evidently attached to the middle turbinate, boggy to touch, covered with a mucopurulent discharge, and anatomic landmarks could not be easily made out. Cocain and adrenalin had little effect on this mass. Transillumination showed a dark area below the left eye and over two-thirds of the internal aspect of the left antrum. Right side negative.

Under 10 per cent cocain in adrenalin I removed some of this mass, which I at first thought to be polypoid in character. Hemorrhage was quite profuse. The anterior nares was packed and the patient was sent home. On subsequent visits we began using snare and biting forceps, but the bleeding was so profuse that little tissue at a time could be removed. After a half dozen sittings we were able to clear the lower part of the nose, and the patient felt so much better that he failed to appear for further treatment. One morning, on November 2, after washing his face he suddenly had a severe left sided nasal hemorrhage and was rushed to the hospital. His nose was packed, and after several hours the patient went home. He now complains of severe pain in antral region and left ear, and says he is getting deaf. External examination shows left eye and cheek more prominent. Internal examination shows lower half still free, but the mass has traveled to the nasopharynx. At this time a small piece from the middle turbinate region was removed and sent to Dr. Allan J. Smith, professor of pathology in the University of Pennsylvania, and he sent me the following report:

"Sections of small fragments submitted show a loose fibrous covering of the ordinary appearance of nasal mucosa, at places with persistence of a stratified columnar epithelial surface, but in the main denuded of such layer. Beneath this in loculi of varying size, separated from each other by fibrous septa, is a glandlike tissue with central spaces filled by a hematoxylin stained mucin. The lining cells of the acinuslike subdivisions of the larger lobules are, however, not of the large, columnar goblet type to be expected in mucous glands, but small cells of the size of an early connective tissue corpuscle, each with a comparatively large deeply staining nucleus, and these cells occur as multiple as well as single lining layers to the spaces. In the fibrous framework of the growth solid cords and isolated islets of the same type of cells are here and there met, indicating an infiltrative tendency in the growth. The vessels are loose in the fibrous framework of the mass, commonly as irregular channels with indefinite walls. The general appearance of the histologic structure would tend to its classification as an infiltrative adenocarcinoma of origin in a mucus secreting gland such as occur in the mucous membrane of the nose, with modification of the cells to the small size above noted. There is one other interpretation open, which, however, the writer is less disposed to consider, viz., that the growth is of endothelial type (endothelial cancer), extending along and lining lymph spaces into which from unknown source mucin has gained access and is retained."

From November 2nd, when I first began to suspect the mass to be malignant, until December 15, the date of his admission to the hospital for radical operation, I used radium in 50 mg. doses directly applied to the mass with slight improvement. These treatments were given at about seven day intervals, and the capsule was retained from three to four hours. Altogether he has had twenty hours' radium treatment. During this time he was given two X-ray treatments by Dr Henry K. Pancoast.

P. M. H.: Has had the common diseases of childhood; no serious illness; no operations.

F. H.: Mother and father living and well; two brothers and four sisters, all living and well. One sister died of T. B.

P. E.: Patient is a well nourished, well developed, white adult male of good muscular and bony development, but short in stature. No abnormalities in gait and station. Skin of good texture; no jaundice or edema. Head normal in size and shape; scalp negative. Eyes: Left eye shows exophthalmus, pupils equal and react to light and accommodation; no nystagmus, ptosis or extraocular palsies. Ears: Negative to external examination, but complains of pain in left ear and deafness. Mouth and teeth negative. Neck: No cervical or thyroid enlargement. Chest: Well formed and expansion good. Abdomen and extremities negative. Radial pulse regular, volume and tension good. Blood pressure, 133—75. Urine examination, December 18, 1919, practically negative outside of a faint trace of albumen. X-ray report by Dr. Henry K. Pancoast shows disease of left antrum, anterior, and posterior ethmoid cells.

Operation, Drs. Muller, Kornblum and Husik: Intratracheal anesthesia. Ligation of external carotid. An incision was made in median upper lip to nose and around left side of nose to root, and straight back below the lower lid. The skin and soft parts were retracted, the whole superior maxilla was sawed through. The eye was not disturbed, as the tissues underneath looked healthy. The middle turbinate was completely removed and ethmoid cells broken down to the sphenoid. There was a large mass of grayish jelly substance in the antrum, which was removed, and, together with the maxilla, was sent to the laboratory for examination. The wound was cauterized, packed and sewed up.

December 19, 1919. Patient feels very miserable; complains of severe sore throat and inability to swallow anything.

December 20, 1919. Very miserable; is given liquids through a tube.

December 25, 1919. Stitches removed from face and neck. Patient is somewhat easier.

December 26, 1919. Complains of severe pain in left ear; drum injected but landmarks not obliterated; complains bitterly about his throat.

December 27, 1919. Developed an acute purulent otitis media. Patient was very uncomfortable until January 5th, when he began to show signs of improvement. He was able to swallow some liquid and said his throat felt somewhat easier.

January 7 and 12. Received X-ray treatments.

Discharged from the hospital on the 14th of January in fairly good shape and reported every other day for treatment to throat and ear. Ear became dry on the 25th, and he is able to swallow some soft diet. There is a good deal of puffiness under the left eye, but this seems to be rather a common condition after such operations.

In March of 1920 I saw a little mass in the region of the superior turbinate, which looked polypoid, and after two applications of radium, 50 mg. four hours each, it disappeared, and to date has not recurred. As time went on, the patient improved physically, was free of all pain, was following the occupation of gardener, could chew fairly well, but had difficulty with solid food.

The report of upper jaw and soft parts sent to Dr. A. J. Smith is as follows:

"Portions of the growth developing en masse in the maxillary sinus came into the general laboratory after the excision of the jaw, the latter being submitted to the laboratory of surgical pathology (Surg. Path., No. 8635), whence a diagnosis of giant cell sarcoma was returned. The writer has examined the latter slides and feels that the diganosis given is not justified, but that the sections obtained in the Surgical Pathology Laboratory show essentially the same features as those here about to be described. They were not made of the bone itself but also from the soft tumor tissue found in the antrum. It is learned that the above diagnosis has been changed.

"The sections here examined included in parts only the intrasinus surface, at places showing a relatively normal stratified columnar ciliated epithelial layer; at other places this is replaced by a single layer of flat epithelium, and at places the surface apparently is completely denuded. Immediately beneath this covering the superficial mucosa is loosely fibrous, more or less edematous, infiltrated by mononuclear and some polynuclear leucocytes, and showing occasional dilated blood capillaries and blood spaces. Beneath this the tumor appears, showing considerable variations on superficial examination but in reality of the same nature generally, with the different appearances occasioned merely by factors of secondary importance. The growth is essentially an adenocarcinoma of the mucous glands of the mucous membrane. At places the glandular growth, while massive, presents well the general structure of glands, and is rather adenomatous than cancerous, no direct infiltration of the interacinous and interlobular fibrous stroma by the epithelium. Here as a rule the stroma is relatively scant and of ordinary fibrocellular character and without special vascularity; but as a rule the epithelium within the basement membrane is excessive and layered. Often, however, a lumen exists containing mucin (sometimes staining normally blue with hematoxylin, but often showing a tendency to take up the pink of the eosin). Where in some portions of the sections the stroma becomes more extensive, excellent epithelial infiltration, cords and islands of definite cancerous arrangements can be found, many fields being rich in this type of growth. Even here, in some of the cords and islets, mucinous collections occur in lumenlike spaces. Elsewhere the stroma shows evident infiltration by homogeneous, eosin staining material, interpreted as probably altered mucin, and at times large areas of such material exist. Into this latter extend lines of epithelium, forming a more or less definite network, the cells at places showing their epithelial character clearly, but in much of such fields being atrophic and resembling more small connective tissue and lymphoid cells. Frequently where the stroma is prominent (as apparently towards the basal attachment of the bits sectioned) there is a rich teleangiectatic plexus of vessels and at places small areas of hemorrhage. In portions showing the frank cancerous character of the growth the epithelial infiltrative cords show syncytiallike masses of the epithelial cells, these being probably expressions of the rapid local growth of the neoplastic tissue, and these probably gave rise to the appearances interpreted as giant cells of a giant cell sarcoma in the Laboratory of Surgical Pathology."

In the spring of 1921, I referred the patient to the Evans Dental School of University of Pennsylvania for an obturator, which Dr. Fox very skillfully made, and which the patient now wears with no discomfort and is able to chew beefsteak.

My last examination, December 27, 1921, shows the patient still free of any recurrence, nose and throat free of crusts; is working, happy and content with life.

Lee M. Hurd in ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, June, 1918, says adenocarcinoma of the nose is rare enough to warrant reporting each case.

Prognosis: In the opinion of the authorities on this subject, the outlook is most hopeless. Thus Carter states: "Prognosis is bad; only recently Bloodgood has stated that not one of the eighteen cases with malignant disease of the nose operated upon at the Johns Hopkins Hospital recovered. There is no authentic case on record where recurrence has not taken place after the operation and often this recurrence has taken place before the patient has left the hospital."

L. D. Alexander, reporting a case of adenocarcinoma with chronologic report in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, March, 1914, says that adenocarcinoma is essentially a disease of the cancerous age, but an early age is possible, as in the two cases he reported, one of 22 and one of 33. My case was 32 years of age.

His case was radically operated upon and no recurrence after six months.

According to Jonathan Wright, adenocarcinoma is the most frequent malignancy of the nose.

Lee M. Hurd, reporting four cases of adenocarcinoma, says case 1 recurred two years and case 3 recurred one year after most extensive radical operation.

Wm. H. Dudley reports a case of adenocarcinoma, with exophthalmos, which lived two years and five months after a radical operation, including the removal of the eye.

Louis Klemptner reports a case in a female 33 years of age, of possible origin in antrum, and extending to ethmoids and middle turbinate, with no recurrence six months after a Denker and ethmoid removal.

L. C. Minor reports a case in a female of 28 years, origin of middle turbinate, after repeated removal and curetage, with no recurrence after one year. Another case in a female of 22 years with no recurrence after exenteration of middle turbinate and ethmoids.

John McCoy reports a case of origin in ethmoid, with extension to sphenoid and frontal, with no recurrence one and a half years after curetage and modified Killian.

As soon as a diagnosis of adenocarcinoma is made by the microscope (clinically it is impossible, because in the beginning it nearly always begins as polyps or polypoid degeneration of the middle turbinate), the best chance for the patient is complete removal, preferably by the external route, and followed up by X-ray and radium.

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XXV.

CHRONIC STENOSIS OF LARYNX AND TRACHEA WITH REPORT OF A CASE TREATED BY LARYNGOSTOMY AND DILATATION.*

By Edward Cecil Sewall, A. B., M. D.,

San Francisco.

Chronic stenosis of larynx and trachea may be divided into functional and organic types.

Chronic functional stenosis occurs in the larynx. It may be caused by panic or by spasm.

Where fear alone is the cause of the dyspnea, the patient is usually the wearer of a tracheal canula. There is nothing pathologic except the psychic state. It is quite common to have difficulty in removing the canula in the inured canula carrier.

Where spasm is the cause, it produces dyspnea by the median position of the cords. It may be caused by the wearing of an intubation tube. Also inflammatory conditions of neighboring parts may produce it.

Chronic organic stenosis may occur in the larynx, trachea or bronchi. Under this rubric is included:

1. Narrowing of the lumen by pressure from without.
2. Paralysis of the abductors.
3. Ankylosis of the arytenoids.
4. Neoplasms.
5. Hyperplastic and cicatricial lesions.

1. Narrowing of Air Passages From Extraneous Pressure.—Such stenosis is of very great importance, chiefly in connection with goiter, and occurs usually in the trachea. In countries where goiter is common, serious involvement of the trachea is found so frequently that an indirect or direct examination of the trachea is properly asked of the laryngologist in all cases before operation. Death from asphyxiation dur-

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ing the operation has been not infrequent in cases where structural parts of the trachea have been caused to atrophy.

2. Paralysis of the abductors is often the beginnnig stage in recurrent laryngeal paralysis. Following Semon's law, the cricoarytenoideus posticus is affected before the lateralis. The paralysis often accompanies tabes, diphtheria, bulbar and other central nervous system disorders. The cords incapable of abduction are drawn toward the middle line by the adductors. Where both sides are affected breath is passed only with difficulty and noisy vibration of the cords accompanies the effort. The condition is a dangerous one and may lead to sudden asphyxiation.

3. Ankylosis of the cricoarytenoid articulations produces a condition that is distinguished with difficulty from that caused by abductor paralysis. It is caused by "rheumatism," or more commonly by perichondritis. As a result there may be actual joint changes or scar formations about the articulation having the same effect. As in bilateral abductor paralysis cords are fixed in the middle line and thus cause stenosis.

Sargnon and Taubert have called the stenoses resulting from median position of the cords "stenoses fonctionelles." Criticism of this nomenclature by Citelli would seem to be well taken, although they clearly distinguish them from psychic conditions. Citelli calls them "stenoses due to the permanent median position of the cords." Excepting for the position of the cords the larynx appears normal.

4. Neoplasms are provocative of obstruction of the air passages, but in general the treatment is the removal of the growth and is outside the scope of this paper. However, recurring multiple papillomata in their nature lead to chronic stenosis and must be considered. They are much less important from this standpoint than formerly. With direct removal, either through the laryngoscope or with the suspension apparatus. they are successfully combated. Radium seems to have a very definite place in the prevention of recurrence.

5. Hyperplastic and cicatricial chronic stenosis are terms describing with fair accuracy a various group of lesions. The causes of such stenoses are diseases or injuries that lead to hyperplastic or cicatricial conditions blocking the air passages. Among these may be mentioned syphilis, tuberculosis,

scleroma, diphtheria, scarlatina, measles, typhoid, whooping cough, and last, and of great importance, trauma.

Injury may cause perichondritis, inflammation and swelling of structural or soft parts. Killian in his report of war wounds of the larynx divides injuries into those of, first, the parts at the entrance to the larynx; second, above the cords; third, in the middle, through or involving the cords; fourth, underneath the cords. A practical point he brings out in this classification is the fact that emphysema of the skin and surrounding parts occurs only in the wounds of the fourth variety. In such cases the cords form a barrier and the air is forced out of the wound.

Decubitus from an improperly fitting intubation tube or the long wearing of a tracheal canula is considered under the head of trauma. It is an interesting observation that in the inflammatory condition associated with diphtheria, laryngeal stenosis occurs where tracheotomy has been done without intubation almost as frequently as where the patient has been intubated. A frequent cause of the stenosis following diphtheria was first described by Köhl as a hypertrophic inflammatory condition of the subglottic mucous membrane.

TREATMENT.

Functional stenosis, when either simple panic or with added spasm, must be treated primarily along educational lines to rid the patient, usually a child, of fear. The long use of a tracheotomy tube makes the patient more or less dependent upon the consequent free breathing. Breathing through a wide canula bears the same relation to breathing through the mouth that breathing through the mouth does to breathing through the nose. We well know the difficulty in teaching some children to be satisfied with nasal breathing after the free, shallow and easy mouth breathing.

The intubation tube may have led to spasm through undue pressure, and it is recommended to try different forms of tube, more narrow in the interglottic region. Tracheotomy will usually relieve the spasm in a short time, where other means have failed.

Narrowing of passages from extraneous pressure. In such cases the treatment has more to do with the alleviation of the condition pressing on the trachea.

Paralysis of the abductors and ankylosis of the cricoarytenoid articulations give rise to fixation, in the middle line, of otherwise normal cords in an otherwise normal larynx. The treatment of both is identical.

Section of the recurrent laryngeal nerve has been done to produce total paralysis and so leave the cords in the cadaveric position and do away with adduction. There is usually room to breathe in bilateral total recurrent paralysis. Both section and resection of the nerve have not given satisfactory results. It was long ago pointed out by Cain that where there was continued action of the adductors without opposition of the abductors that the cords shortened and so retained their median position, even after severance of the recurrent laryngeal nerve.

Intermittent dilatation by sounds has not been satisfactory nor has the sustained pressure of the intubation tube been sufficient to hold the cords permanently aside. O'Dwyer suggested and tried intubation. He was also the first to cut away the cords for abductor paralysis. Hope also tried excision of the cords for this condition. However, Citelli gave impetus to the method when he excised the cords, experimentally, in dogs, especially to determine whether they regenerated as was claimed by Fränkel. Their controversy brought considerable publicity to the subject. Citelli maintained that the cords do not regenerate, though admitting the establishment of fibrous bands for phonation.

Fränkel gave authority to the regeneration theory, and those opposed to Citelli's method maintained that the voice was lost and the cords returned. The truth seems to lie in the middle ground. Certainly new tissue takes the place of the removed cords. It, however, does not take up the amount of room that the cords did. Also the voice is not destroyed, but, though hoarse in quality, is serviceable. So the procedure has a definite place in the treatment. Jackson has reported cases where he removed the cords, eviscerated the larynx through the mouth, with successful outcome.

The removal of cords alone, however, has not been the answer to the question in most hands, and other means of gaining room have been used. Ivanhoff proposed and carried out submucous resection of the arytenoids following laryngofissure. This allowed the cords to be drawn outward and gained room for breathing. Resection of the arytenoids has long been done on horses where paralysis of the recurrent laryngeal nerve has allowed them to become flaccid and be drawn into the larynx on inspiration, causing "roaring."

Sargnon and Taubert added the curettage of the sinuses of Morgagni. The adhesion of the walls, they claimed, would draw the cords outward. It is easy to agree with Citelli that the relief was rather problematical and left much to be desired theoretically and practically. Hansberg and Uffenorde removed the soft parts submucously and practically.

Killian developed the technic as follows: After laryngofissure he dissected up the mucous membrane with underlying elastic tissue, conus elasticus, including that in the subglottic space. Removed the musculus vocalis, also the ventricularis and part of the lateralis. Separated more or less of the arytenoid cartilages. Sewed in place the mucous membranes. Fastened the edges of the laryngofissure wound with the skin, forming a laryngostomy.

Treatment of Hyperplastic and Cicatricial Stenosis.—The remedies that have been proposed and used are numerous and diverse. The severity of the stenosis will suggest the radicality of the methods for relief. In therapeutics the number of remedies is in proportion to the difficulties of the task. The various means for relief of these conditions, however, group themselves under three heads: bloodless or dilating, surgical or cutting, and combinations of the two methods.

The first attempts at relief were made by simple dilatation. This treatment was intermittent and was best exemplified in the use of Schrötter's dilating bougies. These were passed in increasing sizes from the mouth through the larynx. It is almost essential that the larynx be not entirely occluded. Where it requires force to pass the smallest sound grave damage may be done by the formation of a false passage. Cutting through an impermeable stricture from above, except in web formation or where the knife is guided with great certainty under the eye, is dangerous. This intermittent method of dilating has been satisfactory in some cases and is still useful where the stricture is a mild one, easily discouraged.

O'Dwyer with his intubation tubes brought a method of great value which has had tremendous influence upon all the various procedures, surgical and otherwise, used today. While intended, originally, for acute stenosis of the larynx, O'Dwyer soon saw the usefulness of his tubes in chronic conditions. His great skill and the ingenuity of his successors, especially in the American school, have sufficed to conquer by intubation alone unbelievably difficult stenoses. Intubation may be preceded by intermittent dilatation or the use of the expanding dilator until the passages will admit the smallest tube. By their use in successively increasing sizes and with variations as to composition was obtained the ultimate advantage from sustained dilatation, by way of the mouth.

Tracheotomy is considered by this school a disadvantage and like the other operative procedures is only to be employed where intubation has failed in its purpose. Where tracheotomy has been performed greater variety is permitted in the methods of dilatation. It is now possible to dilate the stricture from below. Urethral catheters, or better, the dilators devised by Dr. Lynah, serve this purpose. This intermittent bougieing would correspond to the use of the Schrötter type of sound by mouth. Uchermann has combined the incision of the stricture from below with blunt curved bistoury where necessary before dilatation. This is an easier and safer use of the knife than from above downward.

Making use of the tracheotomy wound, it is now possible to draw down, by means of the "endless cord," different forms of dilating apparatus. The intubation tube may be anchored through the tracheal fistula by some form of "post." Care should be taken that the post and the tube are not both of metal, because corrosion may fix the post and make removal difficult. If the tracheal canula is in situ combinations of all kinds may be made with variations of the obturating part of the apparatus. An intubation tube, one covered with rubber where desirable on account of size or composition, a single piece of rubber tubing, a piece of rubber tubing twice the requisite length doubled upon itself—any one of these suitable to the case may be drawn down into the larynx and fastened by cords to the tracheotomy tube or about the neck. A further development of this same principle is represented by the different forms of tracheotomy tube with dilating plug attached to it by some mechanical device. These tubes have been developed so that they can be separated into units small enough for ready introduction and then brought into position and locked.

Where all the simpler methods fail or where, in one's judgment, they are inadequate, there is fortunately a method which will satisfy reasonable requirements of breathing and phonating in practically every case. Laryngostomy as at present employed is the result of the development of the idea of Herying, worked out principally through the Lyon school, as represented by Sargnon and with the essential aid of Killian.

The surgical details being given in extenso by Jackson, Sargnon, Killian, Moure and others, we will simply sketch the salient features. Briefly the operation consists of

1. Laying Open the Larynx and Trachea.—Jackson's idea of inserting scissors or cutting instruments into the tracheal wound and cutting through all structures, including the skin, in one motion is a great time saver and also favors the wound epithelialization. It leaves a wound edge with successive layers undisturbed by dissection. Where the tracheal wound is lower than the stoma desired, or where irregularly placed, as reported in war wounds particularly, there may be advantage in careful dissection of skin and successive tissue layers.

The excision of the intralaryngeal parts where the stenosis is of the median cord position type has been discussed.

How much of the scar tissue to remove must be determined by the operator in each case. What can be done submucously is certainly advantageous, but the difficulty of the submucous resection of any old inflammatory tissue is familiar to rhinologists. Most cases can be successfully combated by dilatation. Jackson recommends a cut straight through the scar tissue in midline, care being taken not to encroach upon the esophagus.

2. Dilatation of the Cavity and Establishment of Stoma.—Killian's valuable contribution to the operation was the use of soft rubber tubing in increasing sizes for the purpose of dilatation. He showed that the constant pressure of the soft rubber accomplished the purpose with minimum tissue necrosis.

Some tissues are more resistant than others, and the practice is to remove the tubes, watch for inflammatory signs and later replace, gradually increasing sizes of tubes.

Many forms of tube have been used. The combination, whatever it may be, must furnish air as well as pressure. There are three different principles on which the tubes have been built:

First, a simple tracheotomy tube with rubber dilating plug extending upward from its dorsum through the larynx to the level of the base of the epiglottis. This rubber plug or tube is fastened in some manner to the tracheotomy tube. Breathing is through tracheotomy tube, and the patient is unable to phonate during treatment.

Second, a straight hollow tube laid in the cavity and extending from trachea up through the larynx as before. Here the patient can breathe only through the rubber tube, but can phonate during treatment.

Third, Jackson's combination of a tracheotomy tube and the upright rubber tube. The tracheotomy tube has a straight post vertically placed, on the upper end of which is fastened the rubber tube extending upward as in other cases. The added feature of a curved tracheotomy tube connecting through the wound with this straight tube gives the patient ample breathing space and, on closing the external tube with the finger, allows him to phonate.

3. Closure of Stoma.—It is best not to close the external wound until all possibility of further contracture is minimized. The wound causes but little inconvenience, more than offset by the sense of security enjoyed by the patient. However, there is a certain added danger of lung infection from its presence, and so it should be closed as soon as advisable. All cases with tracheal fistulæ have been found to have chronic inflammation of the trachea, often of extreme degree.

If the wound has become in time very narrow, the edges may be freshened and closed by simple suture. Usually the gap is such that some form of plastic is necessary. Again, if not too wide, the turning over of skin flaps will suffice, first with raw surface out and the next drawn over this. There are a number of different plastic methods of value.

Where the stoma is very wide, however, there may not be enough structural value in the skin alone. In such cases transplantation of bone or cartilage into the flap is necessary, according to the methods of Konig, Mangold, Schimmelbusch and others.

STATISTICAL RESULTS OF LARYNGOSTOMY.

Sargnon reviewed 150 cases, 76 per cent cured, 9½ per cent no result, 8½ death. Of his own personal cases, numbering 18, he reports 3 deaths, or 16.6 per cent.

St. Clair Thompson, 36 cases, 6 deaths, 16.6 per cent. Ferreri, 11 cases, all cured, no deaths. Chiari, 20 cases, 7 cured; 2 left his service; the rest still under treatment; no deaths. Jackson, 1915, 18 cases; ultimate recovery in all cases. Bellancioni, 16 cases. Schnabel, 2 cases; war injuries. Joseph Beck, 1915, 3 cases with excellent results; no deaths.

Killian, 1916, 36 cases war injuries; 25 fully cured, 8 still under treatment; good breathing space and more than useful voice; no deaths.

Moure, 1918, 24 cases; 9 cured, 15 under treatment. Brunnings, 1918, 6 cases, all under treatment. Hofer, 1920, 11 cases; favorable results; no deaths.

The statistics show that in recent years the number of deaths from this procedure is almost nil. Also the voice in nearly all cases has been reported as useful, and in many instances excellent. It is usually of hoarse quality and explosive character.

Iwanoff made tracings of the movements of the air from the mouth in normal individuals and patients cured by laryngostomy with dilatation. The normal curve shows a sharp rise during inspiration with but little change in the long sustained curve during phonation.

Those operated upon showed the sharp rise for the inspiration, a short sustained period with another quickly following inspiratory effort. Normal speech requires very little breath, but these patients get up pressure and use it quickly.

The time of treatment varies from a few months, in exceptional cases, to two years or more. The average time of Moure's cases was about one and one-half years.

The following case has been under the care of myself and associate, Dr. J. A. Bacher, who has had equal share in its management:

Referred by Letterman General Hospital, March, 1919. Private A. D. Was struck by shrapnel October 10, 1918.

Projectile entered right side of neck about the level of hyoid bone, passed downward and inward directly through the larynx, making exit on left side below the level of the thyroid cartilage. Tracheotomy performed in France. Has worn tracheotomy tube ever since.

Status praesens: Patient can force air through larynx and so produce strained weak sounds. Can swallow without difficulty, though immediately after injury food passed out through wound. Epiglottis is bent upon itself, displaced laterally and bent over larynx. The structures of the larynx are unrecognizable, either with laryngeal mirror, direct laryngoscope or suspension. Could pass a small probe, two millimeters in diameter, into the trachea.

April 23, 1919. Cocainized larynx and trachea through the tracheal wound, anesthetized skin and deeper parts with one-fourth per cent novocain. Introduced scissors through the tracheal wound, cut through the skin, thyroid and cricoid cartilages, making the tracheal opening continue with the opening into the larynx. Retracted the edge of the wound, being careful not to injure the cartilage.

The structures inside the larynx were unrecognizable. On account of the adhesion it was difficult to hold the walls of the thyroid apart very far. Cut directly backward as near the esophagus as deemed safe. Did not attempt to remove any of the redundant tissue.

Introduced the Jackson laryngostomy tube, number six, and brought down on top of it a piece of rubber tubing, 15 millimeters in diameter, two pieces telescoped, so quite firm, inside diameter six millimeters. This was tied by silk threads to the laryngostomy tube, the upper end at level of top of arytenoids. The wound was then very firmly packed so as to remain open.

Patient was placed in semireclining position in bed and the dressing moistened with bichlorid, frequently, changed by day, and by night, nurses. He had a little difficulty with liquids at first but very soon was handling his food quite easily. He also soon became used to the discomfort of the tube. He left the hospital May 10, 1919. The subsequent history of the case, though tedious, was uneventful.

The wound was held open by gauze until the establishment of the stoma. Laryngostomy tube was removed first, after about two months. There was no evidence, during this time, of any inflammatory trouble from the tube. It was changed again in one month.

September 10, 1919. The laryngostomy tube was taken out, left out for three days and then an extra large bronze intubation tube was inserted through the mouth and fastened in place by a plug, through the tracheotomy wound. The patient at this time might have been considered cured, as the breathing was good, voice fair, and the larynx epithelialized. The remaining treatment has been to guarantee against constriction.

November, 1920. Up to this time patient wore the large bronze intubation tube with post, a month at a time, every other month. Then the tube was permanently removed and the patient was taught "autobougieing." A rubber veterinary catheter 15 millimeters in diameter was cut off at convenient length, passed over a heavy piece of tightly fitting copper wire and bent at a rounded angle. The rounded end was thus easily passed into the trachea through the larynx and, after a few days of instruction, the patient learned to pass the dilator and keep it in place a considerable time.

We advised him to use the dilator daily or at frequent intervals for an indefinite period, as it is quickly done with little inconvenience.

June, 1921. Believing him now safe from recurrence, we closed the stoma. The wound had gradually narrowed so that the skin was simply drawn over the opening, the edges freshened and sutured The closure was complete and satisfactory. There was very little space for raw surface to cause granulation, but if such should form we rely on "autobougieing" to take care of it.

The patient has a very useful voice, produced by vibration of the soft parts at the entrance to the larynx. These tissues can be seen to vibrate on phonation. He can make himself heard at long distances. We tested him at 100 yards and could distinguish his words very readily. His voice has the usual explosive quality. Since leaving our care the patient has married.

Our experience has been limited to this one case and another which is at present in the course of treatment. I presume that always in such cases individual resource leads to some modification of standard methods. We did not follow out the dilatation as advised by some surgeons, especially Moure. He establishes the stoma, and then after that has healed begins his dilatation.

Our case took from the start a sufficiently large tube, and it was not changed for two months. This may not be safe in all cases, but certainly caused no harm in this instance, and we are following the same procedure in the other case which we are treating.

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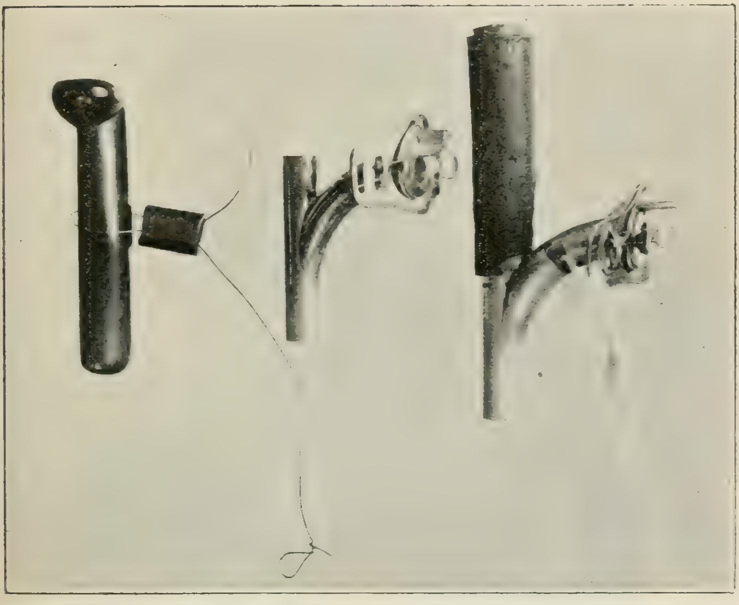
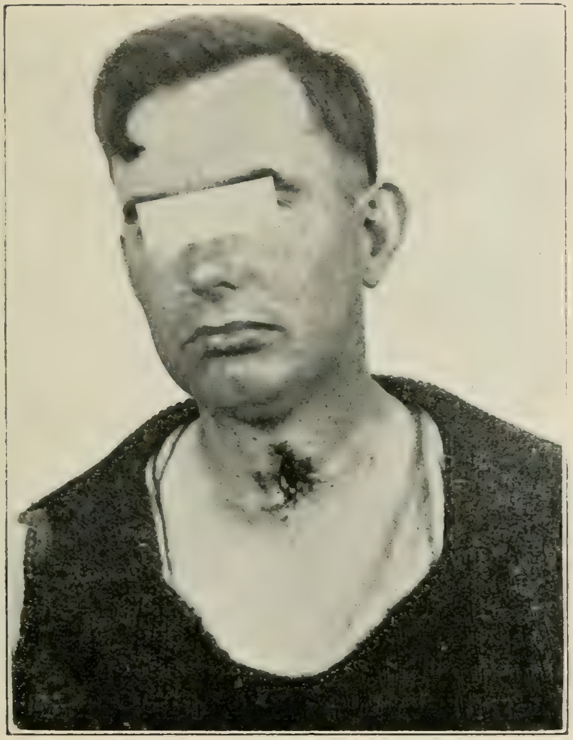
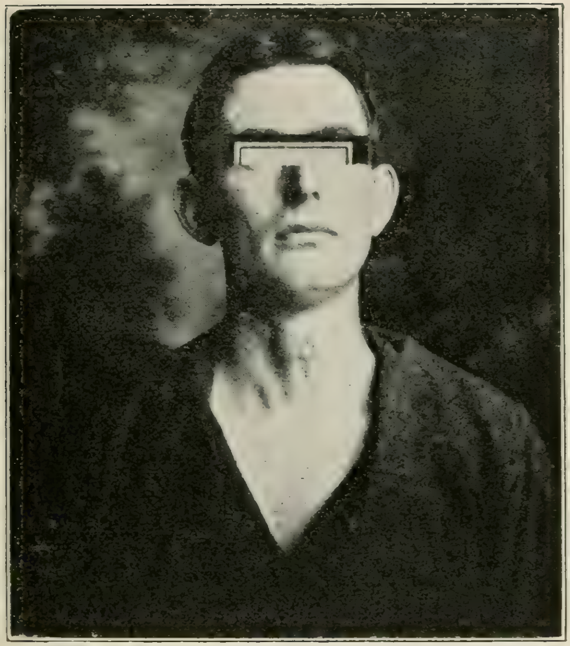


Jackson's tubes and intubation tube with retaining wire.



Patient showing stoma established.



Patient showing wound some months after closure.

XXVI.

THE HISTOPATHOLOGY AND HISTOGENESIS OF BENIGN GROWTHS OF THE NOSE AND ACCESSORY CAVITIES.*

By Harry L. Baum, M. D.,

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It has been the custom of the author of this paper to give more than average attention to the science of pathology in its bearing on otolaryngology, and he believes that this study of a branch of medicine somewhat removed from the usual routine of special work has been the source of much valuable information relative to the specialty and of a better understanding of many cases. As a knowledge of anatomy is essential to proper surgical skill and technic, so in a no less definite way is a knowledge of pathology assential to a proper understanding of diagnosis and treatment. We practice a somewhat limited specialty as otolaryngologists, and it is not too much to ask that we have a working knowledge of the fundamental sciences as applied to that specialty. We may not always be capable of making a histologic diagnosis with the accuracy expected of one making pathology his specialty, and it is not with such an expectation that attention is directed to the science, but in the hope that we may gain a clearer understanding of disease processes and of the changes taking place in the tissues with which we work.

The illustrations which accompany this paper have been limited to microphotographs, thus avoiding the tendency to draw into an illustration what we think should be seen in the section rather than what is actually present there. In this connection it must be kept in mind that no photograph can convey the impression gained from a study of the whole section, but a careful selection of the proper area for reproduction has been made in each case. The photographs were made by the author, using a very simple apparatus, consisting of an ordinary camera box with bellows and ground glass back and without lens, mounted on a vertical support. A Bausch and Lomb balopticon was used as a source of light, interposing a piece of flash opal glass for diffusion while filtration was secured with Wrattan gelatin filters. Orthochromatic plates were used. Exposures were made by turning on and off the balopticon, thus avoiding the use of a shutter.

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*Thesis accepted by the American Laryngological, Rhinological and Otological Society.

The paper makes no particular claim for originality, but is more the result of a careful study of the literature and an endeavor to utilize that study to the best advantage; with an earnest effort to collect a series of specimens typical of the conditions they represent and a careful selection of those proper to be reproduced and presented herewith. Many have been discarded as unworthy, others have been selected in lieu of something more nearly perfect, and still others have perforce been omitted because it has not been the author's good fortune to have met with cases from which to secure specimens.

THE EDEMATOSES.

These, by far the most common pathologic conditions met with in the nose and accessory cavities, have been variously called polypi, edematous polypi, myxomata, fibromyxomata, myxofibromata, soft fibromata and edematous fibromata. Many writers have erroneously adopted terms descriptive of them which have not only resulted in a confusion of terminology but of understanding as to the actual histologic structure of the tissues under consideration. There is nothing in the structure of a nasal polyp which justifies the use of the term myxoma as applied to it, for it contains no myxomatous tissue in its substance. Wright,[49] in 1900, emphasized this point very strongly, and it is also brought out in Delafield and Prudden[16] and by Ewing in his "Neoplastic Diseases,"[18] although many of the older pathologists fail to recognize this differentiation. Expressions such as "fibromyxoma" and "myxomatous" have doubtless been used by many writers with a full knowledge of the laxity of the terms, but they have failed to transmit that knowledge to their readers, with the result that there still exists a deplorable lack of uniformity of understanding and of terminology among rhinologists. The writer pleads guilty to this fault in his own case in the past and feels that a more accurate terminology should be adopted, if not histologically descriptive, at least not actually misleading. To this end the general term "edematoses" will be used in this connection, for the reason that a study of the histology of these growths will reveal that the fundamental underlying change in the tissues is a serous effusion, an edema.

SIMPLE EDEMATOSIS.

To that condition in which there is simply an edema, or serous infiltration, among the fibers of the loose areolar tissue constituting the subepithelial layer of the mucosa and unaccompanied by demonstrable fibrous hyperplasia or marked pathologic change in the tissues themselves, the term "simple edematosis" will be applied. This classification should include such conditions as the apparent hypertrophies due to hay fever and socalled vasomotor rhinitis, many of the more lasting polypoid hypertrophies of the turbinates, resulting, usually, from chronic conditions in the sinuses, and some of the earlier types of pedunculated polypi before they have undergone marked fibrous change. Figs. I, II and III.

EDEMATOUS FIBROMA.

On the other hand, it is true that many of the edematoses will reveal a sufficient amount of fibrous hyperplasia to justify the name of fibroma, and to these will be applied the term "edematous fibroma," as most descriptive. By this is meant a tumor consisting of loosely packed fibrous tissue, the site of serous effusion, containing fibrin threads, leucocytes in the form of the familiar round cell infiltration, blood vessels and at times glandular structure. In short, it may contain any of the constituents of the mucosa from which it springs, to which may be added the signs of subacute or chronic inflammation evidenced by increase in round cell infiltration, increased fibrosis around vessels, dilatation of gland acini and in places marked fibrous tissue hyperplasia. The epithelial changes manifest themselves both as hyperplasia of the normal columnar and in the form of metaplastic change to cuboidal or squamous, the latter usually stratified and frequently hyperplastic. Figs. IV, V and VI.

This classification includes the older forms of turbinate and septal edematoses and the majority of those having their origin in the accessory sinuses. At this point it is only fair to remark that the gradation from the purely edematous conditions to those justifying the use of the term edematous fibroma must be very gradual, and that there must of necessity be a certain elasticity in the application of these terms. It is true, however, that at one extreme there exists the typical simple edematosis and at the other the condition of undoubted fibrous hyperplasia with edema, and although histogenetically they are apparently one and the same process, their evolution from one form into the other justifies the use of slightly different terminology to signify that change.

The histogenesis of this type of nasal neoplasm has been deeply studied by numerous authors, among them Uffenorde and Hajek, whose views are quoted by Marquis,[33] Skillern[47] and others, and it is agreed that their formation is undoubtedly dependent upon some form of irritation or inflammation in the tissues from which they spring. This results primarily in transudation of serum and later in hyperplasia of connective tissue with definite tumor formation, necessitating an accompanying hyperplasia of the epithelial covering to keep pace with the increase in substance of its underlying structures.

PSEUDOCYSTS.

A condition not yet mentioned but frequently occurring in the development of edematous fibromata is that of socalled cyst formation. Although true cyst formation does occasionally occur in the mucous membranes of the nose and accessory sinuses as a result of obstruction of the opening of a mucous gland, the socalled cysts found so frequently in the pedunculated growths in the nose are in reality pseudo cystic in character and are simply dilated connective tissue spaces filled with serum. They are not lined with epithelium as are the true cysts, but with endothelium derived from the connective tissue in which they are formed. Figs. VII, VIII, IX and XI.

ANTRAL, CHOANAL OR NASOPHARYNGEAL POLYPI.

The edematoses in general may have their origin from any portion of the mucosa lining the nose or accessory cavities, but spring most frequently from the ethmoid cells and middle turbinate. When arising from the antrum they have attracted enough attention among rhinologists to have been given a special name, that of antral, choanal or nasopharyngeal polypi. Histologically they are identical with the edematoses as described above, but their peculiar origin and location make them worthy of special consideration in this connection. Since Zuckerkandl in 1893 called attention to their origin they have been a subject of considerable interest, and Woakes, Hajek, St. Clair Thompson and Killian among others have treated the subject thoroughly and have established beyond question the fact or their origin in the antrum. Avellis[4] in 1913 and Ewing[20] in 1918 have reviewed the subject again, and Ewing has taken up that portion of the subject which is germane to this paper. He reports thirty-five cases, of which fifteen were studied histologically, and his observations in this field agree with those of the author[5] expressed in a previously published paper but based on a much smaller number of cases. His conclusions regarding their development differ from mine in one detail, namely, the mode of exit of the polyp from the antrum. It has been my conclusion, from a study of the histology and the gross pathology at operation, that these growths start as either an edematous fibroma or as a cyst of the mucosa from a chronic inflammatory process, probably not suppurative. This fills the antrum, and extrusion through the antral opening occurs as a result of pressure. Plausibility is lent to this theory by the fact that at operation the antrum is usually found to be filled by the growth, that studies of the antral mucosa show an older or more advanced stage of pathologic change in the antral tissues, and because this is the only reasonable, logical conclusion that can be entertained. Ewing maintains that it certainly does not occur in this manner, for the reason that "the antral portion has often been found to be quite small, occupying only a small portion of the posterior part of the cavity." Such is sometimes the case, but since histologic observations show that the antral portion of the growth is always of quite marked fluid content, it is quite logical and reasonable to suppose that after its extrusion through the ostium and its resultant free expansion in the nose there occurs a migration of fluid from the antral tissues into the nasal portion. This would be favored by the greater fibrosis of the older pathologic condition in the antrum and would result in the apparent loss of substance in those tissues. Or, knowing as we do how rapidly under certain conditions it is possible for edematous tissues to shrink from absorption of their fluid contents, is it not logical to suppose that such a simple occurrence might explain the condition in some instances? Figs. X and XI.

PURE FIBROMA.

Although pure fibroma, commonly called hard fibroma or fibroma durum, is exceedingly rare in the nose and accessory cavities, authentic cases have been reported. My search of the literature has revealed but three: one reported by Meigas[37] as fibroma of the septum, but with no histology has been eliminated as having probably been an edematous fibroma, as no other case of pure fibroma of the septum has been reported. Another, not included among the three, is reported by Dabney[15] as having undergone sudden malignant degeneration, which leads to the belief that it was fibrosarcoma from the beginning, as malignant degeneration is questionable in pure fibromata. The original sections in this case may not have reached sarcomatous tissue. Worthington's case,[48] however, appears to be authentic, as is also that of Dempsey,[17] particularly as the latter underwent retrogression and disappeared spontaneously. This growth sprang from the sphenoid and was not limited to the nasal cavities, extending into the nasopharynx as well. Coates' case[13] of multiple fibroma had two parts, the nasal part being separate and distinct from the nasopharyngeal, and for that reason it has been included as being a nasal fibroma. Nasopharyngeal fibromata are more common and although outside the scope of this paper their histology is identical.

Here again we meet the same difficulty in the literature resulting from loose application of descriptive terms, and in view of the fact that most of the benign growths of the nose are, in a sense, fibromata the adoption of a more accurately descriptive term is suggested, that of "pure fibroma."

The histologic structure of pure fibromata consists practically entirely of closely packed fibrous tissue, showing very few nuclei, blood vessels or cellular forms and none of the characteristics of the nasal mucosa. They may be the seat of hyaline change or of calcification. They have their origin in the periosteal connective tissue, and Zandonini[55] ascribes an etiologic significance to inflammatory processes.

PAPILLARY FIBROMA.

This subject cannot be properly treated without reference to Wright's[50] admirable review of the subject, wherein he took issue with Hopmann and others, and showed beyond question that Virchow's terminology of papillary fibroma should be adopted as the descriptive term for these growths, limiting the term to mean a tumor consisting of a profusely proliferated epithelial layer supported by a very small amount of connective tissue with small, poorly developed blood vessels. The epithelial layer has separating it from the connective tissue a delicate basement membrane, demonstrable in most cases, and a few round cells may be found scattered among its fibers. Mucous glands should be absent from the connective tissue stroma, as the tumor develops from the epithelial and subepithelial layers of the mucosa, which contain no glands.

Careless histologic diagnosis has caused great difficulty in the interpretation of the reports of some observers, for the reason that they have often been led to make a diagnosis of papillary fibroma by the papillated appearance of the mucosa caused by infolding from pressure and without the characteristic epithelial proliferation. It is safe to state, however, that the occurrence of papillary fibromata in the nose is rare and their commonest site is on the septum. Arrowsmith,[3] in 1911, reported the thirty-fifth case up to that time, not including the one of Blumenthal[6] reported in 1910, all of which were septal growths. Herzheimer,[28] in 1911, mentioned one case originating in the frontal sinus, Hannemann[26] one originating in the ethmoid and sphenoid, reported in 1912; Anziolotti[2] one in the same year, Johnston[30] one and Brueggemann[8] two in 1913, Callison,[9] one springing from the region of the bulla and middle turbinate in 1916, and Ide,[29] in 1917, a case in which two were present, one springing from the septum and one from the anterior end of the inferior turbinate. In all of these we are justified in concluding that the histologic diagnosis was carefully and properly made, and although all were not located on the septum it is fitting that they should be considered and classed together as papillary fibromata, regardless of their point of origin. Figs. V and XII.

Scarlett[44] calls our attention to another question of interest in this connection, that of the socalled hard and soft varieties of papillary fibromata. This nomenclature has followed the rule that if composed of squamous epithelium the tumor is called hard, if composed of columnar epithelium it is called soft. It is obvious that such a method of terminology is purely arbitrary and should form no part of the histologic diagnosis. It should be replaced by the terms "squamous celled" and "columnar celled," and in Herxheimer's paper[28] he has made use of these terms, calling his case one of "fibroepithelioma papillare cylindrocellulare."

ADENOMA AND ADENOFIBROMA.

Here, as in almost all benign nasal growths, one finds in the literature a confusing lack of agreement as to proper terminology. Ewing's observations[19] suggest that the only true nonmalignant adenoma is that owing its origin to embryonal tissue and adenomatous morphology in general is due to various forms of irritation, functional activity or overgrowth. He recognizes, however, that it is not always possible to make the distinction on morphologic data alone without considering the conditions under which the process develops and its clinical course. Wright and Smith[52] also state that "benign adenoma unassociated with marked evidence of its origin in the stages of chronic inflammation of the nasal mucosa is an exceedingly rare phenomenon." The most interesting case encountered by the author in this connection is that reported by Gault,[21] in 1911, in which the growth is described as filling the whole nasal cavity and attached to the external wall at the ethmoid level and to the roof and necessitating the Moure operation for its removal. The histologic diagnosis of benign adenoma was confirmed by Professors Jacques and Hoche of Nancy, and no evidence of recurrence was noted after five months. Caldera[11] in the same year reported a small tumor growing from the inferior turbinate and the meatus, the histology of which was that of adenoma. These, with Mosher's case[38] and two of Wright's, are the only ones met with by the author that could be classed as benign adenomata, and Mosher, in his article, quotes Hasslauer as reporting but four cases up to 1900. On the other hand, growths partaking of the nature of adenomata but in reality adenofibromata are not so uncommon in the nasal passages. Johnston reports two cases, one in 1909[31] and one in 1913,[32] which he calls fibroadenomata and describes as consisting of masses of fibrous tissue surrounding islands of glands. Allen[1] also reports such a case, which he describes as normal appearing gland tissue embedded in dense fibrous tissue with areas of calcareous degeneration and some areas of new bone formation. Myxomatous degeneration was also evident in places in the fibrous tissue. It is clear that these latter cases fall into the class of inclusions and cannot be considered as true adenomata, being primarily fibrous hyperplasias which include in their mass islands of normal gland structure. Their proper descriptive term, therefore, should be that of "adenofibroma," thus reserving the term "adenoma" for such rare occurrences of this tumor as may present themselves. Fig. XIII.

Wright,[51] in reporting two cases denominated by him benign adenomata and which were practically identical histologically, gives the following description of their appearance microscopically: "Numerous fingerlike processes springing from a common base. These processes are made up of loose edematous fibrous tissue covered by one or two layers of columnar ciliated epithelium; but it is apparent that the epithelial development is a complex one. In no place is there any marked thickening of the layers, but the surface epithelium is seen to communicate and to be continuous with deep indentations and ramifications of it into the underlying stroma. Separate rings of epithelium, ovoid, circular or ramified in shape, and varied in extent, are seen to occupy and make up a large part of the bulk of the growth. Examined with the high power one is immediately struck with the amount of mitotic granules in the epithelial cells. Evidently proliferation is rapidly going on, but not in such a way as to increase markedly the number of layers." This description is so much superior to any of which the author is capable that he quotes it as a substitute, particularly as it has not been his fortune to see sections from a true nasal adenoma.

ANGIOMA AND ANGIOFIBROMA.

These growths, most frequently termed bleeding polypi of the septum, are also subject to much confusion in their terminology, and for several reasons. Primarily, they were thought to be limited to the septum, but Malen[32] in reporting two cases described by him as telangiectatic fibroma and telangiectatic adenomatous fibroma, suggects the term bleeding polypi of the nasal fossæ. Citelli,[12] in reporting five cases, one of which was not situated upon the septum, deals strongly with this part of the subject and advocates discarding the term bleeding polypi of the septum. Matsui[35] reports two cases, both of which he speaks of as bleeding polypi of the inferior turbinate, describing them histologically as consisting of "hyperplasia of connective tissue and increase in vessels, the latter grealty dilated." The cases of Mayer[36] and Callison[10] were both of septal origin, the former defined as a "cavernous angiofibroma" and the latter as a "polypoid hemangioma." Reamer[41] reports one case of angioma of the antrum histologically pronounced benign. Thus it may be seen that the terminology should not include the location, for although most such tumors spring from the septum they may have their origin from other parts of the nose and accessory cavities.

This conclusion, however, does not settle the vexed question of terminology and classification as applied to them. Mosher,[38] referring to Haslauer's statistics, leaves the impression that the preponderance of benign new growths of the nasal cavities are angiomata, as out of 115 growths reported 57 were of that type. Wright and Smith,[53] on the other hand, state that "nasal angioma is a rare form of tumor." This evident disparity is explained by the usual laxity in application of descriptive terms and is probably due to the fact that apparent tumors consisting or simple granulation tissue are comparatively common, especially on the septum. Citelli[12] states: "It will be observed that the etiology of bleeding polypi is the same as that of all tumors, or of any neoformations upon which irritant causes have a marked influence. These irritant causal factors sometimes set up simple or specific granulations, at other times true tumors. These granulations must not be confused with the veritable tumors." Russo[12] also, in reporting five cases under the title of bleeding polypi of the nose, recognizes this differentiation by diagnosing two of his cases histologically as granulation tissue only. He also refers to Citelli's statement to the effect that with the denomination of "bleeding polypi" tumors of various kinds are united in the same class, as, for instance, the granulations and pure angiomata; and quotes Macaigue, Casalie-Brunetti and Denogany, who seem to think that all socalled bleeding polypi should be united in one group and, taking account of their histologic and clinical characteristics, called "fibrovascular benign tumors of the nose." In spite of all this, Russo clings to the old term, thinking a change would only confuse and would solve the problem for no one, but conceding that among the bleeding polypi there should be included neither the pure angiomata nor the granulation tissue masses.

Citelli, in the paper referred to above, goes on to discuss at some length this part of the subject, proposing that "all nasal tumors be classified according to their histologic structure, following the actual rules of pathology," and after a detailed description of his cases, states: "As a matter of fact, the larger part of the nasal tumors to which the designation "bleeding polypi" has been applied are true angiomas: simple angiomas, cavernoma (or more often mixed, simple and cavernous); or mixed angiomas, with connective or myxomatous tissue." Personally, the author can think of no good, scientific reason why these tumors should not be classified according to their histology as granulation tissue masses, angiofibromata, either simple, cavernous or mixed; and pure angiomata, either simple, cavernous or mixed. The term angiomyxomata would be inapplicable here for reasons previously elaborated.

It seems likely, from a study of the description of many of the cases reported by Citelli, Russo and others, that the term "angioma" has in many instances been applied to tumors more properly falling into the present classification of "angiofibromata." There must, of course, be a certain elasticity in the application of these terms and the personal bias and opinion of the observer must be taken into consideration.

Histologically, the granulation tissue masses are simply collections of rapidly growing blood vessels, their walls formed by a single layer of endothelium and supported by a stroma of young connective tissue containing many cells.

The angiofibromata are made up of blood vessels supported and surrounded by fibrous tissue of varying degrees of density and in some instances the site of edematous infiltration. The vessels, particularly those of capillary size, often consist solely of a single layer of endothelium, although in many ot the older tumors with larger vessels the vessel walls may present both fibrous tissue and muscular structure, the latter rarely. The vessels run indiscriminately through the tumor mass so that in sectioning they are cut at various angles. Endothelial proliferation in the extravascular substance is common, particularly in regions showing rapid growth of small vessels, so that in some cases the intervascular structure is largely made up of endothelial outgrowths from the vessel walls. These cells sometimes form themselves into rows as if in an effort to form new vessels. The cavernous types usually show the above picture in some sections, with the addition of dilated blood spaces in others. These spaces frequently show an increase of fibrous tissue in their walls, including proliferating endothelial elements and some fibroblasts. Extravasations of blood into the fibrous tissue stroma are frequently observed. The epithelial covering is usually stratafied and may be either the normal columnar or squamous, the latter usually in the anterior part of the nose from local irritation.

The pure angiomata, practically never met with in the nose, are made up practically entirely of vessels with virtually no fibrous supportive structure and may be either capillary, arterial, venous or cavernous. Their intervascular substance may be made up entirely of endothelial elements and their covering is subject to the same variations as above. Figs. XIV and XV.

ANGIOMYOMA.

Citelli,[12] in the paper referred to above, adds the report of a case of nasal tumor which he defines as an angiomyoma and describes as consisting of capillaries, mainly arterial, which presented a marked hyperplasia of their muscular layer. These vessels were interwoven with and surrounded by bundles of pliable muscular fibers with characteristic elongated nuclei and such fibers enveloped the surface of the tumor like a capsule. It appears that in this instance there occurred in the muscular coat of the vessels a marked hyperplasia resulting in the above histologic findings and as it is the only case of its kind on record is of corresponding interest.

LYMPHANGIOMA.

This condition is so rare in the nose as to receive no more than passing mention in the literature. Johnston[31] mentions a case reported by A. Schmidt, and Wright and Smith[54] refer to one case reported by Hamm and also to one reported by Hajek and Polyak[27] called lymphangiectasis myxoma but which they suspect was a myxosarcoma.

Histologically, lymphangiomata are tumors composed of lymph vessels instead of blood vessels, as in the hemangiomata and their microscopic appearance is very similar to the latter. Never having seen a lymphangioma of the nose, the author will not attempt a description of one.

PAPILLARY HYPERTROPHY OF THE TURBINATES.

This style of hypertrophy is sometimes so marked as to form what is practically a tumor and will be included in this paper for that reason. Although most common on the posterior end of the inferior turbinate, it has also been observed on the anterior end and on the posterior end of the middle turbinate. Its pathologic interest is the same, regardless of its site, and one description of its histology will suffice for all.

The chief factor in the histology of this condition is the marked hyperplasia of the fibrous tissue in the subepithelial layer, usually unaccompanied by edema. This results, of course, in engorgement of venous spaces, in partial obliteration of glands from pressure with dilatation of some of the acini, and in hyperplasia of the epithelial covering. The latter occurs as a direct result of the papillation of the surface produced by fibrous overgrowth, necessitating epithelial proliferation to keep pace with the increase in surface area. Metaplastic changes are also frequently met with in certain places, accounted for by the irritating effects of pressure and disease upon the epithelium. Fig. XVI.

CHONDROMA.

This type of tumor is also of great rarity in the nose, although many such have been described by authors who have doubtless taken septal spurs (ecchondromata) to be such. Sherrill[46] reports a case of enchondroma of the antrum, and Schwerdtfeger[45] also reports a tumor diagnosed as genuine chondroma of nose, frontal sinus, antrum, ethmoid and sphenoid. His histologic findings are quoted: "The section shows the tumor consisting of several small nodules, connected with little tissue containing few blood vessels. The cartilaginous tissue itself contains numerous cartilage cells and some homogeneous matrix negative to elasticin staining and presenting in spots distinctly fibrillary structure." He concludes that these areas show commencing mucous metamorphosis, and if this is true we would here have an example of true myxomatous tissue in the nose. He is led to this conclusion by finding spots wherein the matrix separates into fibers, with evidence of mucous degeneration of the protoplasm of the cartilage cells in the more central portions of the growth. Guggenheim[24] reports a case which he considers benign, with photograph and description, and Moure and Pierre-Nadal[39] report a case of osteochondroma of the nasal fossæ without histology.

OSTEOMA.

A number of cases of osteoma of the nose and accessory cavities have been reported, probably occurring with greater frequency in the sinuses than in the nasal cavities proper. Marx reports three cases,[34] one originating in the anterior ethmoids, one in the frontal and one in the sphenoid. Culbert[14] also reports an osteoma involving multiple structures, including the ethmoid and frontal sinuses, while Boardman and Donovan,[7] reporting a case of osteoma of the frontal sinus, cite Sewell's paper, wherein he states that he found ninety-two cases of osteoma frontalis in the literature. Thus it may be seen that osteomata are not uncommon, but they must not be confused with bony occlusions of the posterior nares such as that reported by Rutland,[43] which are developmental defects and not new growths. Also, they must be differentiated from exostoses, frequent in the nasal cavities, especially on the septum. Histologically they usually consist of dense lamellated bone with few Haversian canals, although they may consist of spongy bone with many vessels and abundant marrow spaces and cells.

LIPOMA.

Goldstein[23] reports the only case of lipoma of the nose or accessory cavities which the author has been able to discover in the literature. His case is described as being a fatty tumor mass in the antrum, the histology of which was reported by Thompson to be as follows: "Connective tissue stroma, holding fat cells in its meshes and enclosed in what appeared to be a fibrous capsule. Small capillaries were found in the mass, and the cells were typical of fat cells of the adult type, although some were larger than those usually found in fatty tissue."

TERATOIDS.

Of this rare condition, a true developmental phenomenon, but few cases appear in the literature, and but two with histologic description have come to the author's notice. Onodi[40] describes his case as consisting of stratified squamous epithelium, hornified, papillated and containing hair follicles and sweat and mucous glands. The stroma was formed of hyaline connective tissue, containing fibroblasts. Haenisch,[25] in 1916, gave virtually the same description of his case, except that it contained muscle, mucous glands and serous glands and adipose tissue. In addition, Gaudier[22] reports a case of cyst of the middle turbinate of dermoid contents and concludes that it was probably congenital. These tumors are not teratomata, being too simple in their construction for that term to be properly applied to them, but the first two evidently fall into the classification of nasal teratoids, the latter being, of course, a dermoid. They are probably of little more than passing interest, as their occurrence is so rare that no one individual is likely ever to encounter one in practice.

CONCLUSION.

From the foregoing it would appear that our classification of nasal growths should be revised and the revision adopted by rhinologists as a whole in order to avoid the confusion resulting from the loose application of terms with indefinite meaning. Thus we should be enabled to differentiate nasal neoplasms according to their histogenetic and pathologic characteristics and not according to their morphology as we are now constrained to do. First, of course, we have to contend with the question, very general among pathologists, as to what constitutes a true neoplasm and whether any of the benign growths in the nose are really neoplastic when histogenetically considered, for so close a relationship exists between the products of inflammation and new growths in the nose that it is at present too difficult a task to differentiate them. It would seem, however, that for practical purposes as rhinologists a classification accepting nasal growths as they are and based on their genetic and histologic characteristics would be sufficient and certainly helpful.

And finally, although many of the conditions mentioned herein are rare, it is the opinion of the author that if more rhinologists had a slight acquaintance with the histopathology of the nose the number of reported cases would be greatly increased. It is not always sufficient to refer specimens to a clinical pathologist, for his knowledge of gross nasal pathology may be insufficient to aid him in the recognition of conditions which, while quite common in general pathologic diagnosis, might be rare and interesting specimens when considered in relation to their origin and causation.

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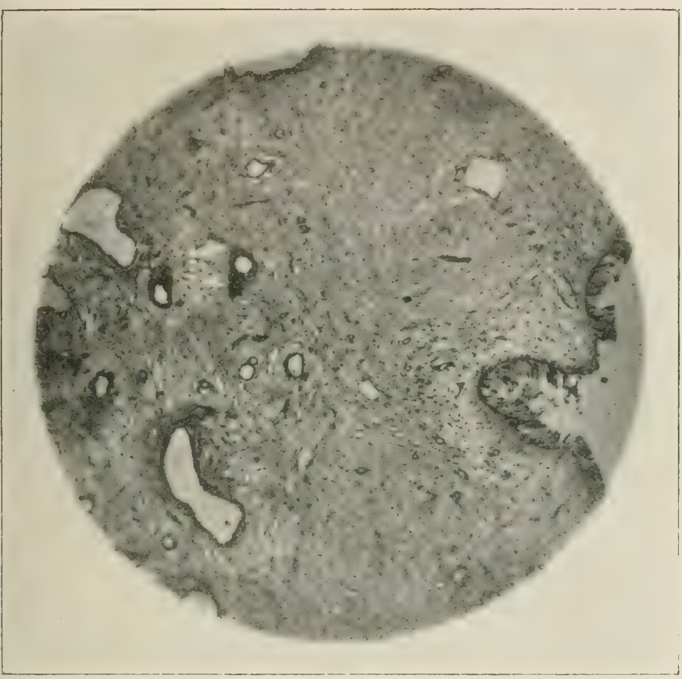

FIGURE I.

Simple Edematosis. Posterior End Inferior Turbinate, Case of Hay Fever.—This section shows no pathologic change whatever, except the marked edematous infiltration of the subepithelial layer, and some increase in round cell infiltration due to diapodesis. Some of the cells are red cells, the majority being small round cells. The normal fibrous tissue of the subepithelial layer is greatly infiltrated with serum with marked distention and great increase in thickness. Epithelium practically normal, glands in some portions of this section show mucous distention in a few acini. Blood spaces apparently normal. Low Power.

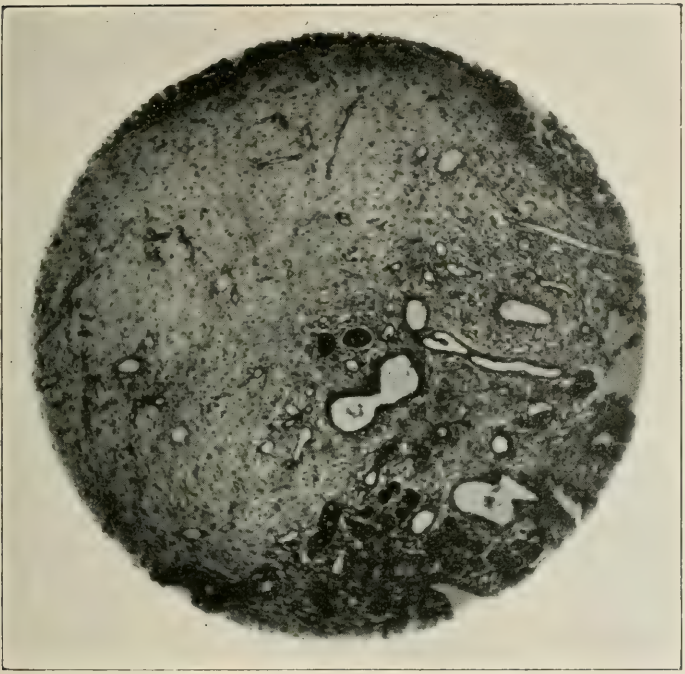

FIGURE II.

Simple Edematosis. Polypoid Middle Turbinate, Chronic Ethmoiditis.—Distention not so acute in this specimen, although it shows no apparent increase in fibrous tissue. Round Cell infiltration apparently normal. A number of dilated gland acini noted, no evidence of vascular congestion. Epithelium practically normal. Low Power.

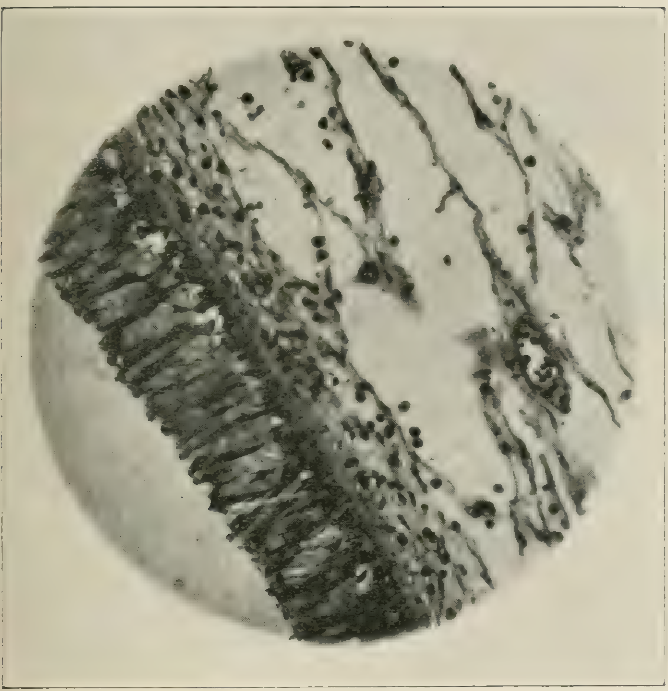

FIGURE III.

Simple Edematosis. Chronic Ethmoiditis, Lining of Cell.—Note marked edema distending fibrillar structure. Normal columnar epithelium, slight increase in round cells, small blood vessel apparently normal. High Power.

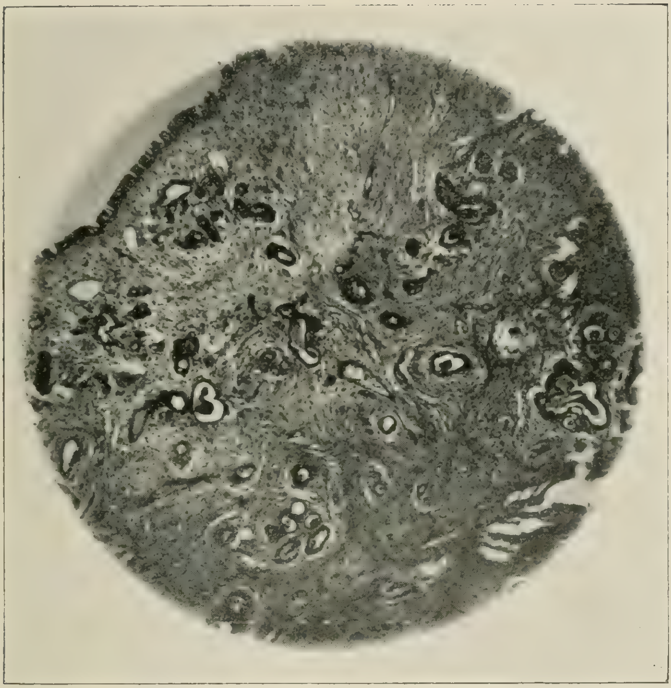

FIGURE IV.

Edematous Fibroma. Middle Turbinate.—This section shows marked fibrous hyperplasia with slight edematous infiltration. Other sections of same growth show more marked infiltration. Mucous glands show considerable dilatation in places, in others normal. Dilated glands show retained secretion and in places marked fibrosis around glands with obliteration of gland structure. Blood vascular spaces over full and at places extravasations have occurred, possibly caused by operation. Fibrosis also marked around vessels in places. Epithelium normal. Low Power.

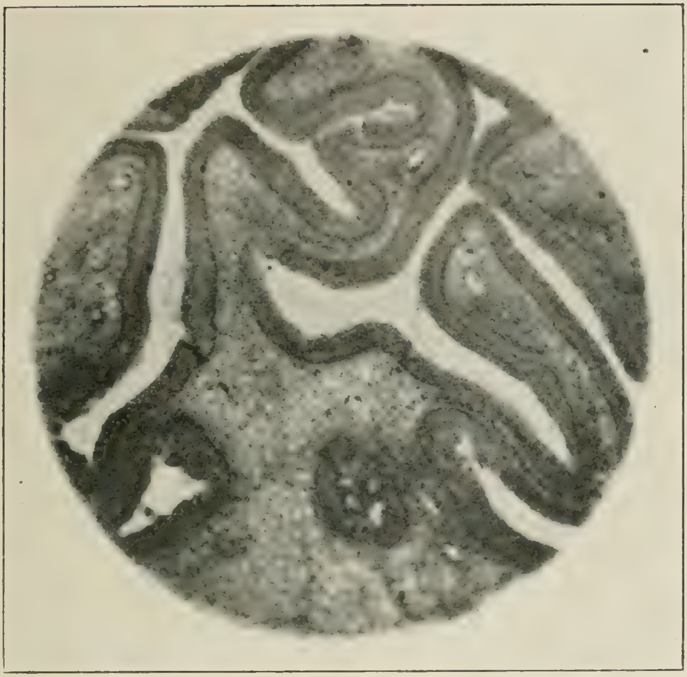

FIGURE V.

Edematous Fibroma. Septum.—Various sections through this growth reveal marked fibrous hyperplasia with but slight edematous infiltration in most places, although same is more marked in others. Increase in usual amount of round cell infiltration and many blood vessels evident. No mucous glands are seen in this growth, probably because of its origin affording fewer such and the effect of fibrous tissue overgrowth in obliterating them. Two interesting changes are noted in this specimen; one that the surface in places shows marked folding from pressure to resemble papillary fibroma but without hyperplasia of epithelium and the other that this section shows metaplasia from normal columnar to stratified squamous epithelium. Other regions of same growth show normal stratified columnar. Low Power.

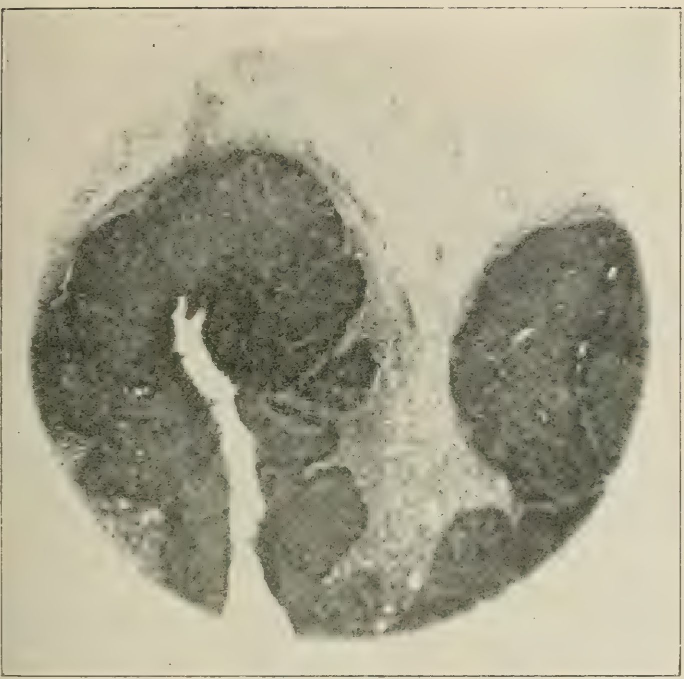

FIGURE VI.

Edematous Fibroma. Epithelial Metaplasia with Hyperplasia.—This section shows strikingly changes in normal epithelium possible in the nose. Although subepithelial structure shows nothing but typical edematous fibrillar tissue, the normal columnar epithelium is replaced by stratified squamous with marked hyperplasia until the section suggests papillary fibroma. This epithelium dips down into the subepithelial stroma but is distinctly separated from it by indefinite basement membrane. Study of the whole section shows that this is simply the epithelial covering of an edematous fibroma, enormously hypertrophied but not making up the body of the growth itself. Low Power.

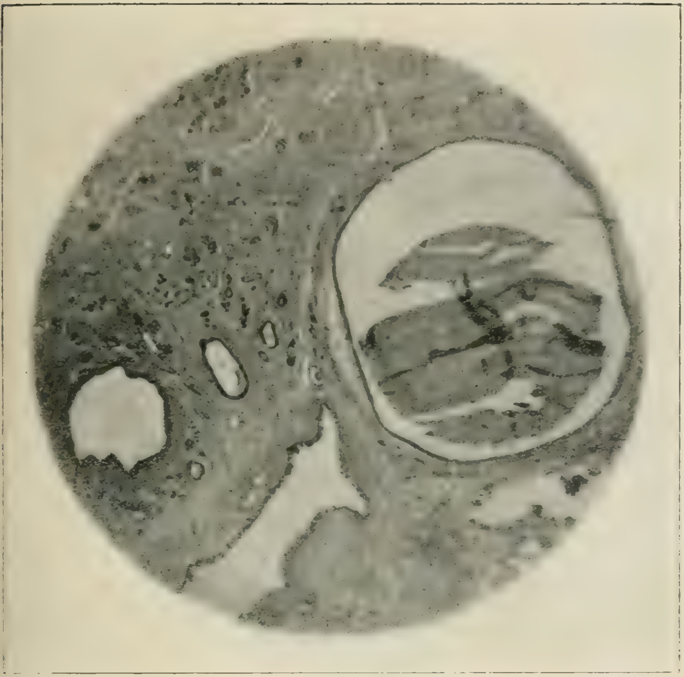

FIGURE VII.

True Cyst Formation.—This section shows the formation of a mucous cyst from obstruction of the opening of mucous gland occurring from fibrous tissue formation in the substance of an edematous fibroma of the middle turbinate. Cyst contains mucous secretion and is lined with glandular epithelium, flattened from pressure of cyst contents. Note marked fibrous tissue hyperplasia around cyst and numerous smaller and one fairly large cyst in same section. Note also surface epithelium dipping down close to cyst wall from folding of growth. Very Low Power.

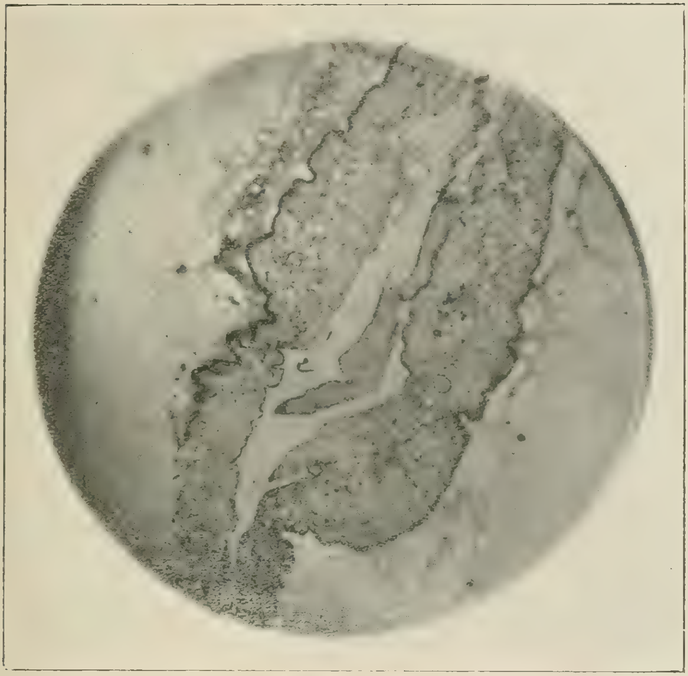

FIGURE VIII.

Pseudocyst of Edematous Fibroma.—This section shows formation of pseudo-cyst from accumulation of fluid in ordinary edematous polyp, so-called. The contained fluid has been coagulated and shrunken from fixing solution. Very Low Power.

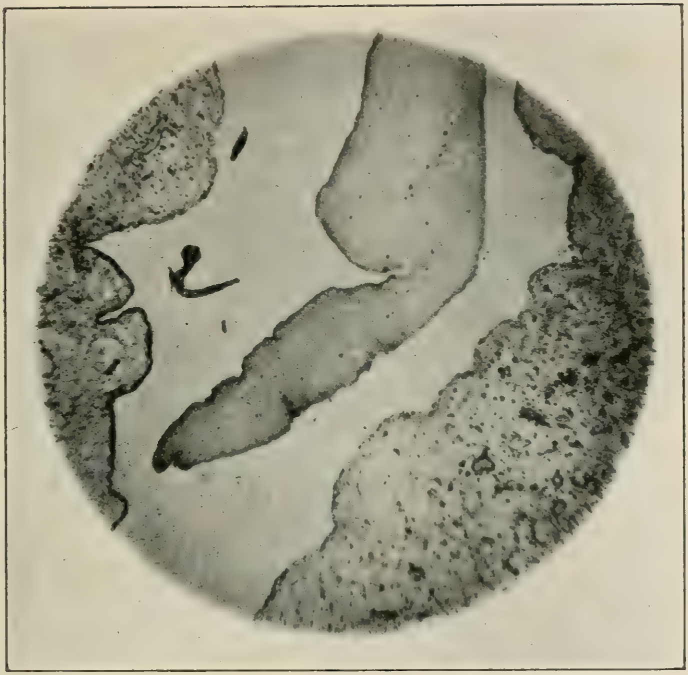

FIGURE IX.

This section taken from above slide to show endothelial lining of pseudocyst and edematous structure of the growth, with its delicate fibrous reticulum, and pseudo-cyst cavity formed in the center from accumulation of fluid in connective tissue space. Note slight cell content of fluid. Low Power.

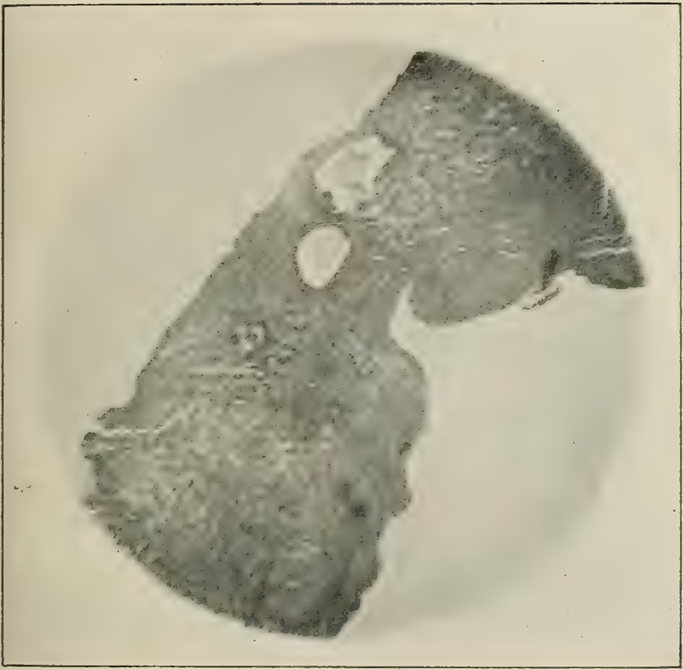

FIGURE X.

Lining Membrane from Antrum in Case of Nasopharyngeal Polypus with Cyst Formation in Antrum.—Note marked fibrous hyperplasia from old condition in antrum. Also beginning cyst formation from dilatation of mucous glands. Also marked round cell infiltration and entire absence of epithelium from pressure. Low Power.

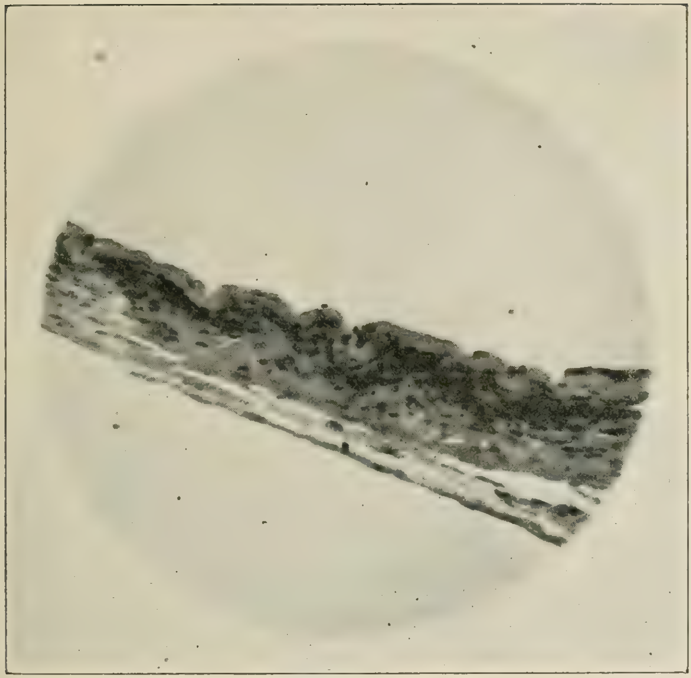

FIGURE XI.

Cyst Wall From Antral Cyst.—This section was taken from wall of antral cyst in same case as above. This cyst was probably originally a true cyst, formed by dilatation of mucous gland as shown in above section in its beginning. All trace of epithelial lining or covering has been lost from pressure, however, and cyst wall consists only of fibrous tissue. High Power.

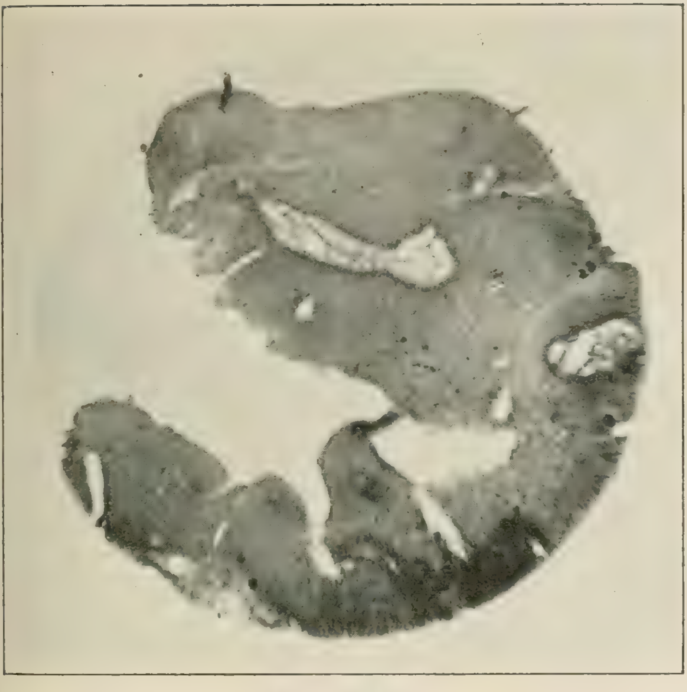

FIGURE XII.

Papillary Fibroma, Turbinate, Squamous Celled.—Section shows marked hyperplasia stratified squamous epithelium, forming papillae with slight fibrous supportive structure, few blood vessels and no mucous glands. Definite basement membrane. Low Power.

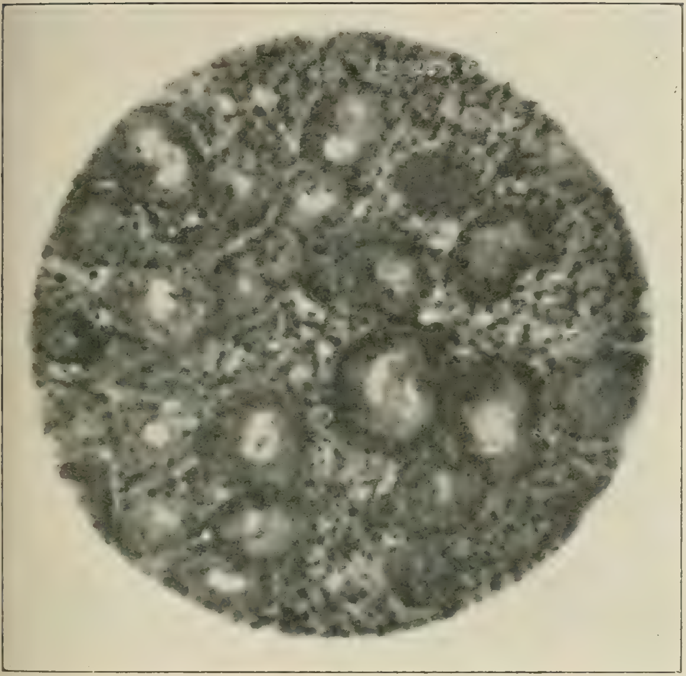

FIGURE XIII.

Adenofibroma, Septum.—Marked hyperplasia fibrous structure, with infiltration and vascular congestion. Most noticeable feature consists of glandular inclusions surrounded by fibrous tissue, these glands apparently normal except for dilatation of acini and greatly increased number. Not an adenoma, simply an inclusion tumor so far as glandular substance is concerned, primarily a fibroma. Edematous in areas. Epithelium, stratified squamous. High Power.

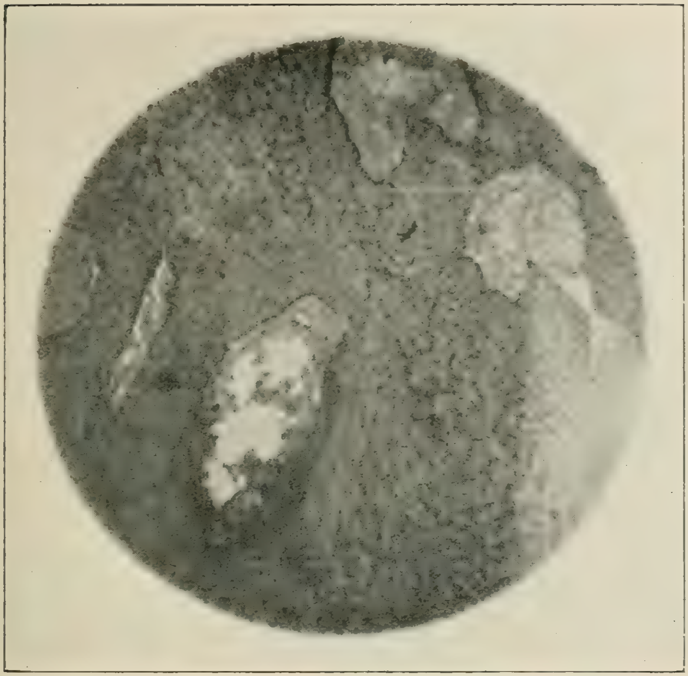

FIGURE XIV.

Cavernous Angiofibroma.—This section shows in other parts numerous capillaries with proliferation of their walls to form new vessels or grow in long strings of endothelial cells often several rows in depth. A comparatively thin stroma of fibrous tissue is present. This field shows most clearly the enormously dilated vessels filled with blood, with endothelial proliferation between the cavernous spaces and fibrous tissue proliferation in their walls including proliferating endothelial elements and some fibroblasts. Some hemorrhage in places. Low Power.

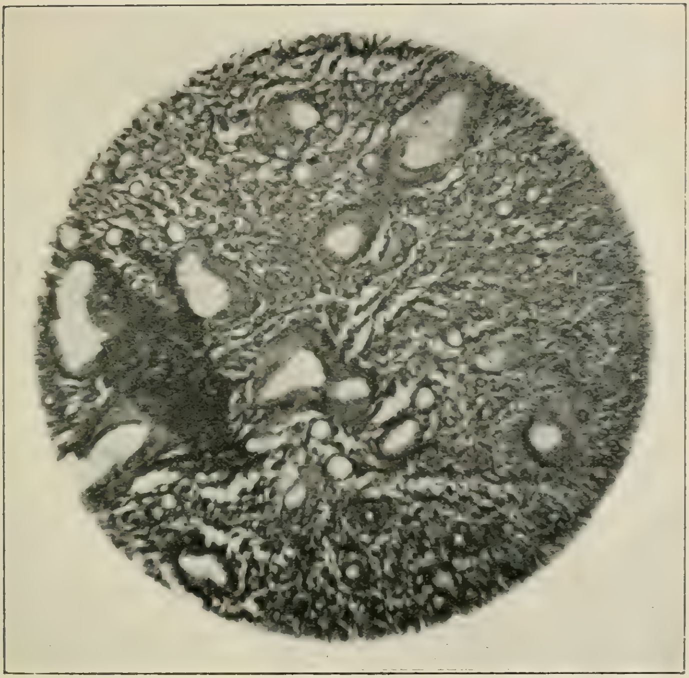

FIGURE XV.

Simple Angiofibroma.—Capillaries and arterioles surrounded and supported by fibrous tissue overgrowth. Some thickening of vessel walls, especially the larger vessels. Some of the smaller vessels show no fibrous tissue in wall whatever, being formed entirely of endothelium. No endothelial hyperplasia is noted in this section, few cellular elements and no hemorrhage. Low Power.

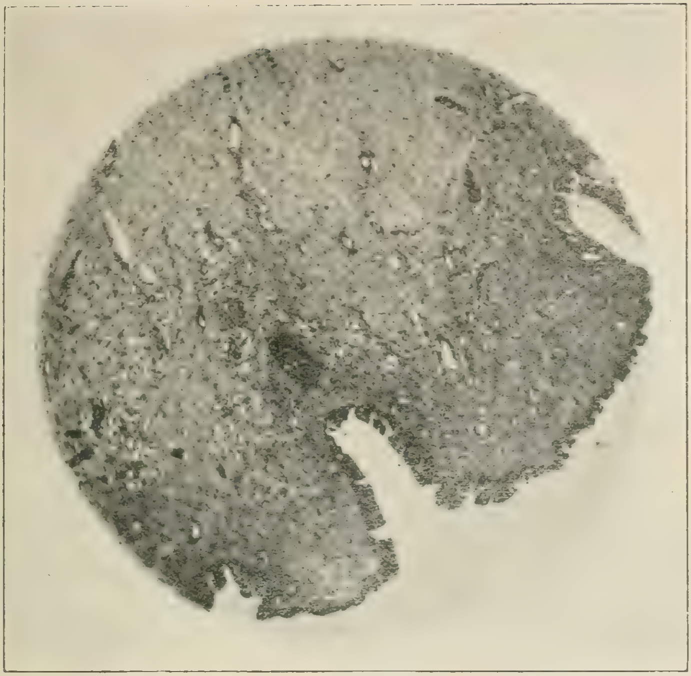

FIGURE XVI.

Papillary Hypertrophy Anterior End Inferior Turbinate.—This section shows marked fibrous tissue overgrowth in subepithelial layer with no edema. Fibrosis evidently old with increase in small blood vessels and apparent lessening in normal number of blood spaces with marked fibrosis around them and around larger vessels. Marked increase in round cell infiltration in subepithelial layer. Mucous glands appear to be normal in number with many dilated acini. The surface of this section shows papillated effect from fibrous tissue hyperplasia. Epithelium practically normal, stratified columnar, following configuration of surface without hyperplasia except as necessary to accommodate to increase in surface area from papillation. Low Power.

XXVII.

NASAL TUBERCULOSIS.

By W. B. Chamberlain, M. D.,

Cleveland.

That nasal tuberculosis is an unusual type of infection is undoubtedly true; that it is far more common than is generally supposed and can be looked for in the experience of almost every rhinologist is the firm belief of the writer. In 1911 he reported two cases before the annual meeting of the Ohio State Medical Association. These cases were subsequently published in the Ohio State Medical Journal and in the Laryngoscope.

During a series of years some ten or twelve cases were seen in dispensary practice, the diagnosis in each case being confirmed by microscopic examination. As accurate records in regard to these cases are not available, they are not embodied in this report. Three cases occurring in private practice, together with full data regarding them, are herewith recorded.

Description.—Confusion still exists in regard to the terms tuberculosis and lupus. Most writers regard the latter term as a relic of the days before the tubercle bacillus was recognized as an etiologic factor common to both processes. Others attribute the presence of the term to the attitude of the dermatologists in applying it to a tubercular lesion of the skin, with which a nasal involvement is often associated.

Killian, in discussing Lennhoff's paper, still insists on the use of the term lupus, inasmuch as the nasal form is always attenuated and characterized by great chronicity. Not infrequently the lupus of the face is simply an extension of a similar process in the nose. This chronic form he considers relatively common, whereas the rapid form, with extensive ulceration, is exceedingly rare—Killian having seen only one case. The vast majority of authors, however, have entirely abandoned the term lupus and speak only of nasal tuberculosis.

There is also great variability in the description of the growth. Onodi divides the process into three groups and speaks of the (1) infiltrative, (2) ulcerative, and (3) proliferative types. Freer adopts a still wider classification, common to many authors, the (1) ulcerative, (2) the solitary tumor form, (3) the diffuse swelling, and the (4) granulating—progressing superficially; while Körner describes the (1) circumscribed tumor, (2) the diffuse infiltration of the membrane, and (3) lupus. The line of distinction between the two latter forms does not seem exactly definite or clear. Possibly the least confusing and simplest classification is that of Zarniko. This classification is adopted by most authors. Zarniko speaks only of nasal tuberculosis, which he classifies into two main groups: (1) the ulcerative, occurring as an end infection in emaciated individuals and complicating a preceding process in lungs, pharynx or larynx, and (2) the tuberculoma or proliferative type, usually regarded as primary, and met with in strong, robust individuals, giving no other evidence of tuberculosis. The divisions made by others he regards merely as variations under the latter group.

Symptoms.—Individuals with nasal tuberculosis as a rule seek relief on account of nasal obstruction. Usually this is the chief, if not the only, complaint. In addition Körner mentions purulent secretion, often accompanied by slight bleeding and a tendency to crust formation. Such cases are accordingly rarely seen early, but only after the process is fairly well advanced. Practically all of my own cases have consulted me on account of nasal obstruction, though more or less secretion has usually accompanied this. Bleeding and crust formation have rarely, if ever, been mentioned. Pain is rare, except in the ulcerative form.

Appearance and Location.—The growth as a rule is situated on the anterior part of the septum and is usually unilateral. It was bilateral on one of my cases only. Out of 164 cases Mygind reports 77 per cent as occurring on the septum. Next in order are the inferior turbinal, the nasal floor and lateral nasal wall. Körner describes syphilis as a process peculiar to the rear of the nose, tuberculosis to the anterior part, the extension to the rear, or bony framework, being secondary. One dispensary case I remember as occurring on the anterior part of the inferior turbinal on one side only, with no involvement of the septum. The opposite side of the nose was normal.

The appearance is that of a graulating mass, mamillated and covered with more or less purulent secretion. On examination with a probe it bleeds fairly readily. The bleeding, however, is not so free as with a malignancy, nor is it as likely to persist. On pushing deeper bare bone is rarely felt. Its presence would suggest syphilis rather than tuberculosis, or possibly an intercurrent syphilis. In addition, the granulations are pale with a very definite demarcation from the surrounding tissue and no surrounding area of inflammation.

Etiology.—Most authors lay stress upon the probable infection from the finger nail in removing crusts from the nasal septum. The possibility of air borne infection should of course be kept in mind. When one considers that the nasal vestibule is the part of the respiratory tract with which infected air first comes in contact infection in this location might be expected more frequently. Factors in its prevention are probably the slight bactericidal action of the nasal secretion, together with the filtering action of the vibrissæ and the flow of the secretion outward. Infection might also occur by extension from the lower part of the respiratory tract, as well as by blood and lymph channels.

Pathology.—Tubercle bacilli are practically never found in the secretion and are often not demonstrable in the tissues. If found at all they are always rare and only discovered after prolonged search. Schultz in describing my first two cases speaks of "the number of tubercle bacilli as being surprisingly small. It required prolonged search through a large number of sections to find an occasional isolated, acid and alcohol fast bacillus. From the nature of the lesion and the numerical relationship of the bacilli, one gains the impression that the bacilli are not only not numerous but also not very virulent." Of the histologic picture Schultz says: "Of the various elements which may be present in the tuberculous process—tubercle formation with giant cells, caseation, lymphoid infiltration and proliferation of fixed tissues—infiltration with lymphocytes predominates and characterizes the lesion, whereas caseation is less marked in both cases than one usually finds in such an amount of tuberculous granulation tissue."

Zarniko's description is so concise that I give it verbatim. "The growth consists histologically of a diffuse heaping up of round cells in which single typical giant celled tubercles are scattered. At times the tubercular structure is not so evident. Then the existing giant cells of Langhans lie free in the granulation tissue. Tubercle bacilli are so rare that they frequently are not to be found, even after prolonged search. The tendency to caseation is slight. The covering consists usually of stratified pavement epithelium, which here and there sends processes into the depths of the tissue."

Differential Diagnosis.—Nasal tuberculosis is most likely to be confused with syphilis, though Onodi performed a radical operation on a case supposedly malignant, which he found subsequently to be tuberculous. Zarniko calls attention to the following differences:

1. Tuberculosis involves primarily the cartilage—syphilis the bone.

2. Fetor is rare in tuberculosis—common in syphilis.

3. In tuberculosis there is no inflammation of surrounding parts—marked inflammation of surrounding parts in syphilis.

4. In tuberculosis headache and involvement of the trigeminus rare—common in syphilis.

5. Tuberculosis is a slow process—syphilis a rapid one. In addition may be mentioned the Wassermann reaction, the administration of potassium iodid, the injection of tuberculin, as well as the histologic examination. The general examination will often prove of value as demonstrating evidences of tuberculosis or syphilis in other parts of the body. Zarniko calls attention to the fact, however, that we may find syphilis in a tubercular subject, or vice versa.

Treatment.—This is entirely surgical and consists in removal of the granulating mass with snare, punch forceps or the sharp curette. When located on the septum Körner advises the resection of the involved area by a wide incision through the septum, leaving of course a large septal perforation. Subsequently the areas are treated with lactic acid on account of its selective action, or the use of superheated air as devised by Holländer.

The prognosis is, as a rule, favorable, leading to early healing and cicatrization, though some writers speak of frequent recurrences. All of my own cases, with one exception, have yielded readily to this treatment with no recurrences. One case has been followed for a period of ten years. One case of the ulcerative type was lost sight of, so that the ultimate outcome is uncertain. This was the only case where pain was complained of. The growth has been removed with forceps and curette well into the normal mucosa, the cartilage forming the base of the area. Healing has been prompt, with no septal perforation.

REPORT OF CASES.

Case 1.—Jessie W., college junior, was first examined in October, 1914, on account of obstruction of the left nose. There was much thick secretion on both sides. High up on the cartilaginous septum on the right was a small punched out ulcer two millimeters in diameter, surrounded by an area of granulation tissue. On the left was found a somewhat larger and more superficial ulcer, with a larger amount of granulation tissue. The Wassermann was negative. There was no reaction following the administration of potassium iodid. General examination showed an old healed tuberculous process at the right apex. Two weeks after the first examination the mass on the left was removed over an area two centimeters in diameter and completely down to the cartilage. This tissue was submitted to Dr. David Marine, who pronounced it tuberculosis, tubercle bacilli being demonstrated. The lesion on the left was completely cicatrized eight weeks after operation. Eight months later there was slight recurrence on the left and a fair sized mass on the right. Both masses were removed. Four weeks later there was complete healing. This case was followed some ten months later. At that time there had been no recurrence.

Case 2.—J. M. S., physician, was first seen in February, 1921, complaining of nasal obstruction on the left. A submucous resection had been performed some years previously. Examination revealed some mucopurulent secretion, beneath which was a cauliflower, granulating mass two centimeters in diameter. There was slight bleeding on probing, but no bone was felt. General health was excellent and no history of syphilis. Wassermann not taken. The mass was removed and submitted to Dr. Howard T. Karsner, professor of pathology in Western Reserve University. His report is as follows: Specimen consists of several small masses, some of which are covered by a stratified squamous epithelium which in some places shows elongated papillæ, which, however, do not appear to be other than proliferation due to inflammation. The tissue is made up of a connective tissue network, in some places dense and hyalin, and in other places loosely arranged. Scattered about are numerous tubercles with centrally disposed epithelioid cells surrounded by lymphocytes. In about half the instances typical Langhans giant cells are found. The density of arrangement of some of the connective tissue indicates that the process is chronic rather than acute. Diagnosis: Chronic miliary tuberculosis.

Case 3.—Mrs. A., aged 38, was examined in February, 1921, on account of left sided nasal obstruction. This was found to be due to a pale granulating mass situated along the anterior septum. There was little or no shrinking with cocain and adrenalin, and no bleeding. No bare bone was felt. This mass was removed, but unfortunately was lost. One month later a granulating mass along the anterior part of the inferior turbinal, together with a necrotic mass posteriorly, was removed. This was submitted to Dr. H. Goldblatt, resident pathologist at Lakeside Hospital, who reported the following: Histologic description—Sections of tissue from inferior turbinal. The superficial epithelium is of the stratified columnar type. These epithelial cells are very markedly swollen, and their cytoplasm is either clear or vacuolated. The epithelium varies in thickness and over a large portion of the tissue is deficient. In the subepithelial tissue there are several large and small areas of necrosis surrounded by giant cells of the foreign body and Langhan's type, by epithelioid cells and by lymphoid cells. Where there is no necrosis there is rather marked vascularity and marked diffuse infiltration by lymphoid cells, eosinophiles, plasma cells and endothelial cells. In some areas there are present large collections of epithelioid cells surrounded by giant cells. Throughout the tissue there is some diffuse fibrosis. In the absence of a positive Wassermann, and in the absence of other signs of lues, this could undoubtedly be considered as a tuberculous process, but vascularization and fibrosis, however, make the diagnosis of tuberculosis in the absence of this information rather difficult. Special stains for tubercle bacilli will be made and an additional report sent. Diagnosis: Granuloma, probably tuberculous.

XXVIII.

THE PROBLEM OF MIDDLE EAR MECHANICS.

CHAPTER II.

BY A. G. POHLMAN, M. D.,

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ST. LOUIS.

The first chapter of this paper introduced some of the facts of comparative anatomy and physiology of the middle ear complex and their relation to the inner ear. The mechanics of the mass displacement theories was discussed by taking the recent theory of Wrightson as a type. Seven leading questions were asked and answered in some little detail. It may be well to summarize the conclusions reached in Chapter I by repeating these questions and giving a brief answer to each.

I. Do the members of the ossicular chain act as an intensifying press which decreases the amount of excursion at the stapedial footplate in response to the drum membrane displacements occasioned by sound vibrations?

Reactions in the drum membrane and ossicular chain to actual plus and minus pressures in the air of the external canal and middle ear have been demonstrated. The drum membrane displacements arising from pressure fluctuations and from contraction and relaxation of the M. tensor tympani and the chain of events which appears to follow these displacements in the dead animal have been applied directly to the conditions as they obtain in the living animal and to the problem of sound transmission. This has been done without reference to the power, the frequency and the distance through which sound pulses of minimum or optimum amplitudes operate against an aperiodic apparatus. The experiments of Koehler will be considered in detail in Chapter III. The work to be done lies beyond the force applied under the conditions imposed and with the type of apparatus which must respond. The sound transmission apparatus gears down the drum membrane movement, but this function is not related to sound transmission. The double lever system of the mammal makes an altered mass response in relation to sound transmission physically inconceivable.

II. Is the antagonistic action of the M. tensor tympani and the M. stapedius responsible for a balance in the ossicular chain?

Wrightson has followed the conception of the muscle function as proposed by Politzer without considering the recent work of Mangold and of Kato. If we are to assume that the ossicular chain is balanced by the antagonism of the two muscles, then these muscles must be reciprocally innervated. The contraction phase of one muscle must be accompanied by a relaxation phase in the other. It has, however, been demonstrated that the M. stapedius may contract independent of either contraction or relaxation of the M. tensor tympani. It has also been shown that the M. stapedius will hold its contracted condition on a stimulation of the M. tensor tympani. Further, in birds, the single M. tensor tympani represents a physiologic peculiarity in that we find a striped skeletal muscle without an opponent.

The writer has described the function of the passive traction system of elastic ligaments associated with the incus as positive return agents against which the contraction of the M. tensor tympani operates. The statement that the M. tensor tympani and M. stapedius are opponents cannot be accepted. These two muscles do not balance the ossicular chain in the mammals any more than does the single M. tensor tympani in birds balance the columellar apparatus.

III. Is the intratympanic pressure equal to that of the atmospheric?

Wrightson assumes that intratympanic pressure is normally equal to that of the atmosphere, and yet he furnishes a reflex mechanism which is supposed to maintain this relation. He does not consider the possibility of tubal closure increasing the pressure within the middle ear, nor does he consider the possibility of air absorption creating a negative pressure in this cavity.

IV. Is the otic capsule filled with liquid and membranes to be regarded as an inelastic container apart from the fenestra vestibuli and fenestra cochleæ, and what relation have these two windows in preserving the balance of perilymphatic pressure which Wrightson assumes is that of the atmosphere?

The otic capsule may not be regarded as an inelastic container; the liquid and membranes may not be considered incompressible; and the two windows do not have the function of regulating the perilymphatic pressure in the manner described. The relation of atmospheric pressure to that of the perilymph is an essential feature in the Wrightson theory but it is not a fact.

V. May the membrana basilaris be considered a transformer of vertical into transverse vibrational displacements at the hair cells because of the relation of stapedial footplate and the membrana tympani secundaria, and because the route through the basilar membrane offers less resistance than the liquid displacement through the helicotrema?

This inferred functional relation rests upon two premises, neither of which can be demonstrated. First, if the membrana tympani secundaria shows no response to the contractions of the M. tensor tympani, it is logical to assume no response will occur to any mass displacements in the drum membrane brought about by sound vibrations. Second, the amount of resistance at the helicotrema is computed with the idea that there are no other pressure regulating factors save a compensation movement at the membrana tympani secundaria. Other regulating factors have been noted. The Wrightson basilar membrane response is therefore a hypothetic solution for the hypothetic mechanic assumption which in itself is not in accord with the facts.

VI. In what manner does the Wrightson piston action of the basilar membrane occur when the sound vibrations are transmitted through the temporal bone to the inner ear?

Wrightson considers the otic capsule inelastic to the forces which drive the stapedial footplate into the oval window. In spite of this he contends that the condensations and rarefactions in the petrosum as a result of bone transmitted sound vibrations may give rise to a pressure response in the perilymph which results in a thrusting of both window areas into the tympanic cavity. We are therefore confronted throughout the Wrightson theory with an involved series of elastic and inelastic containers and a compressible and incompressible liquid membrane filler in all possible combinations. This appears essential to make the theory conform with the observed data.

VII. Wherein does the comparative anatomy show a confirmation of the Wrightson theory?

The weakness of the confirmatory evidence proposed by Keith consists in the strength which his evidence gives to the contention that the structure of any organ is dependent on its ancestral pattern. The similarity of inner ear structure in various animals in no way confirms the Wrightson theory any more than it does a dozen others. It has been mentioned that the theory does not meet the requirements in birds as well as it does in mammals. This is particularly true in those forms which have a membrana tympani secundaria which is practically extratympanic in position. The appeal to the comparative anatomy by Keith practically avoids every consideration of the mechanics of the Wrightson theory save the possibility of the basilar membrane response. Keith offers no detailed account of wherein the comparative anatomy of the middle ear region may help us to understand the obscure relations in the higher forms.

The displacement theories of hearing are based on the premise of an indirect activation of the end organ. They assume that the ear mechanism operates something like the phonograph which, as Wrightson has stated, engraves a molar displacement. The drum membrane accordingly swings in and out and transfers this movement through the ossicles to the perilymph. The shiftings in the perilymph are transferred to the end organ through displacement of the membrana basilaris or the membrana tectoria, or both, and are compensated at the round window. We have presented objections to this conception and have stated there is no evidence that the restorative forces in this aperiodic apparatus are sufficient to produce the end result. We therefore present the mechanics of the second possibility which assumes that part of the energy striking the drum membrane is transmitted as a sound pulse into the ossicular chain and is discharged as a sound pulse into the perilymph. The theory supposes that all mass movements which arise in the drum membrane, due either to excessive sounds or to other causes, are damped out before they reach the perilymph.

The presentation of another method of sound transmission may perhaps appeal to the reader as new. As a matter of fact, however, it is so old that it has been practically forgotten. Carlisle[9] in 1805 makes the following statement: "It does not appear that any degree of motion ever subsists between the ossiculæ auditus as wholes which bears any relation to the peculiar vibrations of sound; but rather that the different motions of these bones only affect the membrana tympani and alter the degrees of contact in their articulation so as to influence the intensities of violent impulses; sounds of less impetus, not requiring such modulation, are transmitted through the conducting series by vibrations of the integrant parts of these bones, unaccompanied by muscular action. This reasoning is suggested by the columella in aves and amphibia."

Johannes Mueller,[28] in his Handbuch der Physiologie in 1840, devotes some space to careful experiments on the behavior of sound vibrations in reference to the problem of molecular transmission. Several interesting statements are made concerning the transmission of sound in terrestrial vertebrates. "Sound vibrations in air undergo a pronounced damping in being transmitted into water but pass readily into water through a tense membrane applied to its surface." "Sound vibrations in air pass readily into water if the tense membrane is applied to a short solid body which alone touches the water." "A small solid body which is applied to the water through a window in the container and is attached movably through a membraneous border transmits sound vibrations into the water more readily than other solid parts. The transmission is, however, rendered more effective if the small movable solid is attached to the central area of a tense membrane which is flanked on both sides by air." "Sound vibrations pass more readily from a membrane to a freely movable solid to water than from membrane to air to membrane into the water. Comparing the conditions with those of the tympanum, greater intensity in transmission will occur through drum membrane, ossicular chain, oval window to the liquid of the inner ear than through the air of the tympanum through the membrane of the round window."

The fountain of knowledge in so far as it pertains specifically to the middle and inner ear behavior in relation to sound is none too limpid. It took the writer six years to precipitate any very tangible information out of a voluminous and detailed bibliography. Accordingly it may be reasonably safe to assume that any other individual of average intelligence might experience similar difficulties. The effort made in this paper is to present the facts and the viewpoint clearly. The suggestion of the simple telephone as a solution for the drum membrane and ossicular chain complex in the transmission of sound must therefore be considered somewhat in detail. The object is to describe the reactions to sound vibrations—not merely to name them. We may agree with Zimmermann when he says that the drum membrane and ossicular chain are not directly related to sound transmission. It is therefore necessary to analyze the behavior of the energy coming through the air to the drum membrane and the route by which it travels to the inner ear.

VIII. May some of the energy of the sound pulse impinging upon the drum membrane be transmitted to the liquid of the inner ear as a sound pulse and without mass displacement in the apparatus?

Let us consider our sound transmitting system in the form of the primitive wooden stethoscope—with a wide flared end applied to the patient and the smaller flared end applied to the external auditory canal of the observer. The sound vibrations arising within the patient are readily transferred to the wood and pass to the opposite end because wood is an excellent medium for sound transmission. The vibration in the wood will tend to remain within it rather than to pass into the surrounding air. It will appear at first sight that the efficiency of this form of stethoscope might be increased if the interval between the aural end and the drum membrane were filled with water. This, however, is not the case, as may be readily illustrated by the insulation which a drop of water affords to the discharge of the vibrations of a tuning fork when it intervenes between the stem and the resonator. It may also be demonstrated by filling the external auditory canal with water and applying the stem of the vibrating fork directly to the liquid. The experiments performed by Mueller, who filled the external canal with macerated paper, may be duplicated by placing the tip of the finger in the external canal. We may apply the base of the vibrating fork to the metacarpophalangeal joint. The sound intensity through this source is considerably greater than if the fork is applied to the mastoid or the cartilage of the ear with the finger in place. That the finger furnishes an excellent medium for the transmission of sound vibrations may be observed in placing the tip of the finger on the resonator and by applying the fork to the knuckle. The response in the resonator is directly affected by the amount of pressure on the finger tip. In other words, the bone and cartilage transmission is excellent, but the connective tissue acts in the same manner as soft rubber. Sound transmission is rendered more efficient through compression of both connective tissue and rubber.

The tip of the obturating finger discharges the sound vibration into the air of the external canal. It also confines the pressure to this area by not allowing an escape through the external meatus. It will appear from this alone that the drum membrane affords an excellent catchment area area for sound vibrations in air. It must also be noted that a large amount of the energy of the sound vibrations entering the external canal is normally damped out through reflexion and through escape of energy toward the outside. This point will be considered in detail later.

We must therefore consider the string telephone. We will indicate the behavior of energy schematically, in a diaphragm (drum membrane) supported by a bony ring (annulus), connected through a bone-cartilagerod (sound transmission system) to a hole in the bony container (otic capsule) which includes a liquid (perilymph). It must also be remembered that the rod is insulated from its window by the interposition of elastic connective tissue. The scheme presented in the accompanying figure 2 indicates the behavior of the energy, and the entire surface of the drum membrane will necessarily be affected by the wave front striking upon it.

The sound wave (SW1) strikes upon the surface of the diaphragm (D), which is supported by a ring of bone. The diaphragm separates the air-containing middle ear from the external auditory canal (E.A.C.). The sound wave impingeing upon the drum membrane will be reflected back and may therefore be represented in two quantities: (a) showing the back reflection and (b) indicating the quantity entering the substance of the membrane. The quantity (b) will tend to remain within the diaphragm or will pass through it as (d). The (d) quantity has a reflection back at the inner surface of the membrane owing to a decided change in medium (d1). The remainder of (d) will pass on through the air of the tympanic cavity to strike against the otic capsule (O.C.). Its course will be interrupted at every change of medium: mucous membrane (d2), connective tissue, periosteum, bone (d3), and finally periosteum (d4) before it enters the perilymph (d5). The sound vibrations entering the bone may perhaps also follow the lamellar grain because the transmission is more rapid in this direction. The amount of energy transmitted to the perilymph will be materially reduced. It is unlikely that any appreciable amount of the energy enters the perilymph through this route under normal conditions with drum membrane intact.

The quantity (c) will travel out toward the periphery, where a certain amount would be reflected back (c1), and a certain amount will enter the annulus (c2).

The quantity (e), made up not only of a part of (b) but also added to by c1 and d1, will pass to the sound transmission element. Part will reflect back at the area of the change in medium (e1), and part will follow the bone-cartilage rod to the oval window. Here it will be more or less insulated from a discharge into the petrosum, not only in the reflection back at (e2), but because the elastic tissue is a relatively poor transmitter unless it is under tension (see later). The quantity (e3) will therefore arrive at the perilymph (P1).

The quantity (SW2) indicates the behavior of the energy striking the drum membrane from the cavity of the middle ear. Apart from a difference in the behavior of the quantity d and a which have changed places, the results will be about the same. The small insert figure merely emphasizes the mechanical relation of the second opening in the otic capsule (round window) to a large mass displacement in the diaphragm. The round window is not necessarily associated with the transmission of sound vibrations.

It must be remembered in considering this scheme that we are regarding the transmission of the energy. It may be likened to the transmission of sound vibrations from a diaphragm along a tense iron wire to another diaphragm. Compare this figure (2) with that reproduced from Wrightson's book (Fig. 1), in Chapter I.

Disregarding the quantity (c), it will be seen that the bone-cartilage system affords a more efficient means for discharge of the energy within the diaphragm than air on either side. The force passing through as (d) not only undergoes more numerous reflections but must pass through a more efficient transmitter, bone (O.C.), into a less efficient one of the perilymph. This is naturally disregarding the various surfaces in the (d) route which may be multiplied in the mucous membrane on the diaphragm and the mucous membrane and the submucous tissues of the otic capsule in bringing the relations over the animal condition. It must also be noted that the physical character of the membrane in comparing periphery and center will be altered by the relative tension exerted. All other conditions being the same, the energy will travel in the direction of greatest tension, whether the diaphragm be pushed out by the rod or pulled in. Let us again liken the condition to the string telephone. The diaphragm tension must be greatest at its application to the transmission system. This tension is indicated in the diaphragm (D) by stippling. It will also be observed that the efficiency of the apparatus is not dependent to a great extent on the original direction of the sound wave. The same amplitude (S.W2) applied to the inner surface of the diaphragm, disregarding the dead area of the attachment of the transmission system, will give the same result as (S.W1).

This diagrammatic representation of transmitting the sound vibration answers the argument of Zimmermann[43]. Zimmermann contends that we disregard the drum membrane and ossicular chain as transmitters of sound. He does not explain the decreased hearing capacity in individuals with lacerated drum membranes.

IX. On the basis of the string telephone transmission system, in what manner are sound vibrations influenced in their passage from the air through the middle ear complex?

The suggestion of the molecular nature of sound transmission was first noted in detail by Johannes Mueller. It has recently been specifically championed by Boenninghaus[6] in his interpretation of the relations in the water-adapted mammal, the whale. This mammal keeps the regions of the external auditory canal submerged and presents a form in which the effects of mass movements in the water upon the perilymphatic space may be eliminated. The range of acuity of hearing in the whale is, after all, a doubtful quantity in spite of the well developed end organ, the large auditory nerve and the extensive crossing of the axones from the cochlear nucleus (corpus trapezoideum). The whale displays a notable departure from the usual middle ear picture in mammals in that the external auditory canal is obliterated. The small rudiment of the lumen near the drum membrane is filled with desquamated epithelium; the ossicles are heavy and united; the stapedial footplate is joined to the massive petrosum through synchondrosis; and the fenestra cochleæ is rudimentary. It may also be mentioned that the petrosum is markedly separated from the remaining head bones through spongy bone containing air cells which are continuous with the cavum tympani.

Boenninghaus contends that the entire system is directly opposed to mass movements in so far as sound transmission is concerned. He also believes that hearing may only result in terms of molecular vibration, to which the writer agrees. However, our conception of what the whale actually does hear and a conception of what sounds might arise within the water are necessarily somewhat limited. At any rate, the development of the internal auditory system may not be considered a safe criterion. However, the proposal that the condition represents one of adapted efficiency to transmit vibrations from the water to the perilymph is open to contention on the basis of the diaphragm-rod theory. In particular this is true when the inferred functional relations in the whale are carried over, as Boenninghaus has done, to the remainder of the mammals. Boenninghaus believes that the stapedial footplate is directed through contraction of the M. stapedius, so that vibrations are discharged toward different points of impingement on the vestibular wall. This accommodation proposition is entirely beyond a physical and physiologic possibility.

The writer is in absolute agreement with Boenninghaus' interpretation that the end organ is affected only by molecular vibrations and not by mass movements. The functional adaptation placed by Boenninghaus on the whale's apparatus is, however, not in keeping with the physical requirements. Whatever a whale does hear, it probably hears through bone transmission other than the ossicles. It lacks an efficient drum membrane, and the synchondrosis of the stapes will tend to diffuse the energy transmitted into the petrosum rather than concentrate it on the perilymph. The adaptation is therefore not one which makes use of the old ossicle chain. It is rather one which eliminates the ossicle chain from the function of sound transmission. The massive development and persistence of ossicles are therefore no better criteria of a functional requirement than the development of the internal mechanism. Let us remember the preliminary paragraph in this article and the statement that "a persistence of phylogenetic structure and phylogenetic function must therefore be carefully borne in mind. Both structure and function may persist in a system without reference to its apparent office at the present time and may be entirely overlooked because the 'adaptations' appear to be complete and the present relation of structure to function too plainly obvious."

The physical requirements for sound transmission may be fulfilled with a proper catchment membrane and a stethoscope-like cartilage-bone transmitter which is insulated from the petrosum by an annular ligament so that it may transmit its energy to the perilymph. There is still another point to be explained. This is in reference to Zimmermann's work in which he deduces that the drum membrane and sound transmission system do not function for sound propagation but merely act as dampers. Zimmermann,[43] while he agrees with Boenninghaus on the molecular nature of the sound transmission, disagrees violently with the latter in practically every other detail. The interesting statement of Zimmermann that sound vibrations pass directly through the middle ear and through the otic capsule to the perilymph is in keeping with the writer's diagrammatic representation of energy behavior (see Fig. 2d quantity). So far we are in agreement. In so far as a consideration of this as an efficient route, let alone *the* efficient route, is concerned, we are absolutely in opposition. Let us therefore look into a matter which practically all of the exponents of mass movement have completely disregarded and which Zimmermann has not considered. The general physics of both propositions is the same and must be clearly understood.

The efficiency of catchment of energy in the diaphragm will depend not only on its physical character, its shape, its tension and its thickness, but also on its position in reference to the wave front striking upon it. It should therefore be placed parallel to the wave front so that its thickness is in line with the displacing force applied to it. Let us consider this disregarded point in some little detail, because no drum membrane in nature is placed in this position. The position of the binaural hearing with the attention directed toward the source of the sound, as determined by eyes and nose, places the external auditory canals roughly at right angles to the line from the source of the sound. Further, no animal possessing an external auditory canal has a drum membrane placed at right angles to the axis of the canal. It is always found in a very oblique position. Let us assume a drum membrane placed at 45° to the axis of the canal. The efficiency of this membrane toward stopping the energy in wave front entering and traveling parallel to the long axis of the canal would be markedly reduced to what it would be if the membrane was at 90°. However, not only is this canal markedly curved and crooked but the orifice of the canal lies almost parallel to the direction of the sound wave from its source. The result of this condition is that not only a mass movement force be tremendously interfered with but necessarily a molecular one as well. This may be stated in another way: the amplitude of the original sound pulse at the external canal must very considerably exceed the amplitude of drum membrane reactions.

The laboratory of nature, particularly on the basis of natural and sexual selection in reference to sound, has been subject to great experiments. Yet we find no animal in which the drum membrane is placed at right angles to the direction of the sound. Can one imagine a phonographic registration resulting through a horn the diameter of the diaphragm? Suppose the horn were turned about on itself like a reproducing phonograph with a small external opening, and with the singer singing across this opening and not into it. Clearly, as Boenninghaus has well stated, the phonograph and the ear mechanism are different in their reactions.

Immediately two mechanical factors suggest themselves: First, if the most efficient position of the catchment membrane is at right angles to the line of thrust, then if the membrane is placed obliquely the amount of surface offered to the thrust must be markedly increased; second, the reactions in the membrane will not only be influenced by its size and position but also by its relative size to that of the external opening in the auditory canal. It is found that in all higher forms the drum membrane is placed obliquely to the axis of the external auditory canal. Its area computed at a right angle position is also considerably greater than that of the available external opening. In other words, the megaphone of a phonograph recorder is reversed. The big end of the horn is at the diaphragm while the small end is roughly at right angles to the line of the sound source.

The phonograph represents a type of instrument in which mass movements resulting from sound vibrations are efficiently recorded. The recording phonograph operates through the mass reactions in a diaphragm which responds to intense sound vibrations. The apparatus of the middle ear, however, operates under conditions where no phonographic registration of sound vibrations will occur. The entire apparatus of the middle ear is not physically constructed for the rapid response essential to efficient mass reactions. Further, when sound vibrations of sufficient intensity are exhibited to give rise to mass responses, such responses are either inhibited or entirely "damped out."

Zimmermann's contention that the drum membrane and ossicles are not the means of transmitting sound vibration might perhaps be thought to be in harmony with the foregoing statement, but this is not the case. Zimmermann is basing his contention on the assumption that sound vibrations pass through the drum membrane, practically as if it were non-existent, and that the sound vibrations are transmitted through the otic capsule to the perilymph. He is therefore not concerned with the efficiency or the inefficiency of the drum membrane. He merely disregards it in favor of a better and therefore more efficient route. This conception is somewhat like that proposed by Kleinschmidt.[22] [23]

It appears from Zimmermann's work that although he holds the molecular propagation of sound vibrations, he misconceives the behavior of the sound pulse in reference to the physical character of the media through which it passes. Moreover, he has not considered the working of a string telephone. Inasmuch as this last point has been thoroughly considered, it is essential that we analyze the efficiency of bone transmission in the sense employed by Zimmermann.

It was shown in Fig. 2 that the (d) factor was reduced through a failure of a more rapid transmitter (drum membrane) to yield its energy readily to the less rapid transmitter (air). It was also pointed out that a reflection back occurred at every step in the change of medium dependent on its coefficient of density/elasticity. Let us eliminate the drum membrane and bring the full energy of the sound wave against the medial wall of the otic capsule. The course of the sound pulse through mucous membrane, connective tissue, periosteum, bone, finally periosteum and perilymphatic membrane will involve an innumerable series of reflections and dampings. As soon as the sound vibrations enter a rigid material like bone they may tend to remain within it through reflection back of the insulating connective tissue. They may tend, moreover, to follow the lamellar grain rather than pass across it. A great amount of energy therefore may be translated from a longitudinal to a transverse direction because sound vibrations behave in this manner in solids but not in liquids. The thickness and intrinsic structure of the petrosum may also limit the amount of energy discharge into the perilymph. The reason that individuals with stapedial fixation and normal end organ are deaf is not explained by Zimmermann. We may deduce that they are deaf because amplitudes of sufficient intensity and low pitch may not be introduced into the external canal under normal conditions to affect the perilymph through the petrosum. This, at least, does not appear possible with the drum membrane intact.

The drum membrane, by reason of its position and structure, interferes with the sound vibrations passing through it from the external auditory canal. It does so because of the change in medium through which the energy must travel and because of the marked reflection back from its surface, and from that of the tortuous canal itself (see later).

The problem of the damping effect upon sound vibrations in their transmission to the perilymph is a complicated one. It is well to indicate that at least four phases must be considered: first, the effect of the shape and position of the external auditory canal; second, the damping effect of the drum membrane to sound vibrations passing through it into the cavity of the middle ear; third, the effect of drum tension on the sound transmission apparatus; and fourth, the damping effect of muscle contraction.

The photographic registration of sound pulses has been markedly improved within the past few years. In particular, the behavior of the sound pulse in reference to its passage through various tubes and membranes, by Foley of Indiana University,[14] are well worth a close study by otologists. The following four photographs, presented through the courtesy of Professor Foley, shows the silhouette of a sound pulse which may be likened to serial photographs of a distending soap bubble. In the later, a limiting membrane is formed by a film of soap and water. In these photographs a limiting zone is created by the refraction of light caused by condensation and rarefaction in the air. The limiting zone of condensation (dark) and rarefaction (light) is therefore a criterion of the enormous amplitude in this short wave length produced by an electric spark. The sound pulse in the first three photographs, taken at about 1/1,000,000th sec., shows the behavior of the distending pulse in its passage through four kinds of schematic tubes: A straight cylinder (a); a megaphone with convex walls, small opening in and large opening out (b); a megaphone with the large opening toward the sound source (c); and a crooked tube (d). The reader will remember the dark band is in advance of the light band and indicates the direction of transmission. (Figs. 3, 4 and 5.)

In following the progress of the pulse through the straight tube (a) it will be noted that but little reflection occurs. There is but little decrease in the width of the band and practically no "bowing" in the wave front. Compare this with the megaphone (b). Again we find little evidence of reflection but a marked "bowing" of the wave front and a consequent dilution of amplitude due to the "side-slip" or diffraction. This "bowing" of the wave front may be readily seen when the position of the band at the edge of the megaphone is compared with that of the sound pulse in the straight tube. Compare this with the megaphone (c) and observe the enormous reflection back from the curved sides. Practically all the energy is reversed except that in the small area traveling on through the end of the megaphone. This small area compares favorably both in position and intensity with that of the corresponding point in the straight tube.

It is interesting to note the behavior of the sound pulse traveling through the crooked tube. Here an enormous dilution in the original amplitude takes place through reflection of the energy at the convex sides of the tube. The position of the wave at the opening corresponds to that in the straight tube (i. e., the velocity of the sound is dependent on the medium through which it is traveling) in spite of the irregular course. The energy of the sound pulse as indicated in the width of the band has been materially damped out.

These photographs demonstrate the damping effect of a crooked tube very convincingly. In likening the behavior to the conditions in the external auditory canal, it must be remembered that in the photograph we are dealing with a single pulse of great amplitude and extremely short wave length in which the wave front enters the opening directly. Whereas, in the case of the external ear, we are dealing with much smaller amplitudes, much greater wave lengths and a wave front which enters the canal almost entirely through diffraction. It is not unlikely that a diagrammatic canal may be constructed and placed in the form and position of the human external ear and external auditory canal. If the same amplitudes were applied as in these figures, the amount of energy arriving at the lateral opening indicated in Fig. 5 in the crooked tube would be so greatly reduced it might even fail to register by this method. And in any event, were a delicate membrane placed across this opening no photographic registration of a sound pulse passing through the membrane might be observed (see Fig. 6). The diffraction of the sound vibrations into the external auditory canal is undoubtedly supplemented through reflection from the external ear, as Kato's experiments seem to indicate (see later).

We may carry over this information to the report of Burnett's[8] experiments which were undertaken in Helmholtz's laboratory. The sound vibrations in this work are carried over from the source by means of a rubber tube 1 m. long. The aural end of this tube was connected with a glass tube fixed in the external auditory canal. "The position of the glass tube in the external auditory meatus has a great influence on the experiments. When the tube is directed downwards and forwards, the experiments were almost invariably successful, but in any other position they may be partially or entirely unsatisfactory. For in the former position the sound waves strike directly against the membrana tympani and hammer, whereas in any other position they are forced against the sides of the auditory canal and are deflected or destroyed before they reach their destination."

It must be remarked in this type of experiment the external auditory canal was closed to the outside and the escape of back reflected energy through the external meatus was limited. The amplitudes of the sound vibrations conveyed in this manner are probably beyond normal intensities. The writer experienced no difficulty in hearing a low pitch fork attached to the end of a meter long rubber tube under similar conditions, even when the tube itself was twisted. It also appears mass drum membrane reactions arise only when the route to the membrane was rendered straight so that a direct impingement of intense sound vibrations might take place.

Nagel and Samajloff[30] made similar observations by means of the clever arrangement of connecting the middle ear region in such a way that it was so interposed in the feed to a sensitive flame. "It was found necessary to maintain an open pathway through which the sound vibrations might pass to the drum membrane, and the tube, tied in the external auditory canal, was placed under tension so that the canal could be rendered straight." "We applied a vibrating fork upon a cranial area corresponding to the head of the malleus and observed pronounced reactions in the flame. This was more pronounced when the external auditory canal was closed. This may be considered new evidence of the incorrectness of the Harless notion that the interpretation of the Weber and Wheatstone experiments (that a tuning fork is heard more loudly through the teeth when the external auditory canal is closed) might be regarded as a deception or error in judgment. The experiment further demonstrated that vibrations in the bone are carried over to the air of the external auditory canal and from this to the drum membrane."

Nagel and Samajloff also observed that similar reactions took place in the sensitive flame to vocal and bone transmitted sounds. It is not unlikely that the Harless interpretation is not a correct one. The transmission of vibrations through the finger in the external auditory canal with the fork applied to the knuckle illustrates this in a simple manner. It is also probable that bone transmitted sound vibrations normally pass through the ossicular chain. On the other hand, the experiments of Nagel and Samajloff were performed under abnormal conditions. They probably were dealing with abnormal intensities in a dead animal, and the explosion waves which arise in speaking or whispering into a tube were not ruled out. The detailed article of Harless on "Hearing" is worthy of careful consideration by any student of this subject. Harless was a prominent exponent of the theory of molecular transmission and even diagrammed the "centering" of the energy upon the footplate of the stapes.

Dennert[11] is also an exponent of the molecular transmission of sound vibrations through the ossicular chain. He makes an interesting series of objections to the experimental evidences on the mass movement reactions. We take the liberty of quoting from his work because his article has not received merited attention:

"An attempt has been made to confirm the theory of mass movements in the sound transmission apparatus by means of ear models and through the findings in normal and pathologic specimens. These experiments are indeed germain because they indicate the interrelation of the entire intratympanic apparatus including the labyrinth contents in toto. A movement of the apparatus, including the contents of the labyrinth, has been established with the manometer as a result of gross mechanical causes, such as the experiment of E. Weber, in which pressure was exerted upon the apparatus through the finger; those of von Helmholtz, in displacements of the manubrium mallei in a model constructed by him; in the registration of plus and minus pressures; and under the influence of very powerful sound vibrations. But these observations do not demonstrate that similar mass movements of the sound transmission apparatus, including the labyrinth contents, actually occur under normal conditions of hearing, because under normal circumstances we are dealing with extremely minute power factors. The observations on the phonograph have been regarded as an important substantiation of the mass movement theory, and it may not be denied that these observations are apparently in accord with the ideas of mass movements in the sound transmission as applied to the ear. But here also, as has already been mentioned in regard to the experimental investigations on models and preparations much greater forces are required, and the demonstration is one which shows a tremendous superiority of the ear over the phonograph in relation to sound vibrations of small intensity. According to Morak, whispered numbers may be correctly understood at a distance of 89 m., but they are not registered by the phonograph even when spoken directly into the megaphone. The vast superiority of the ear over the phonograph may still better be demonstrated in the quantitative method of testing hearing devised by myself in which, with a low pitch fork, a certain number of vibrations are permitted to impinge upon the drum membrane in a certain unit of time. While one of my forks with a vibrational duration of 30 sec. may be picked up as intermittent in passing it by the external canal three or four times a second, practically throughout its entire duration, the phonograph, in spite of its diaphragm and its lever mechanism, fails to record this same fork when vibrated continuously deep in the megaphone. We may therefore regard the observations on the phonograph as analogous to the above mentioned observations on models and specimens and as gross mechanical processes of sound transmission, while the molecular transmission to the ear is a more ideal, and much more delicately refined process connected with minute forces in the sound vibrations." This may also be regarded as an answer to the Wrightson contention that the inner ear must operate under similar conditions to those under which the phonograph registers the molar movements of its diaphragm.

The damping effect of the drum membrane on the sound vibrations impinging upon it and being transmitted through it into the air of the middle ear also deserves mention. According to Zimmermann, the membrane offers little or no resistance. Professor Foley obligingly made a photograph of the reflection back of a sound pulse projected against a delicate collodion membrane probably not one-fifth the thickness of a human ear drum. (Fig. 6.) This membrane was supported by a wire holder which appears as a heavy dark band. The amount of reflection back is enormous, and if the reproduction of the figure is successful only a delicate line will complete the circle on the far side of the wire holder. This may be taken as evidence of two things: First, the actual damping effect of a membrane, and second, the very small amount of energy which is required to stimulate the inner ear through the sound transmission system. If one can overhear the conversation in an adjoining room through a closed door it is probably safe to assume that not 1 per cent of the energy in the first room passes into the second through the door and walls.

The damping effect of the drum membrane will therefore mean on the basis of the diaphragm-rod theory that only a very limited amount of the energy transmitted through the external canal will pass into the drum membrane. This will necessarily limit the amount of transmission of sound vibrations to the perilymph because of the small and inefficient catchment area.

It has been demonstrated that the position and character of the external auditory canal has a decided damping effect on sound vibrations. These vibrations must enter through diffraction or a sideslip in the wave front of practically 90°. It was also shown that a large amount of the original energy entering the external canal is reflected away from the drum membrane area. It has been indicated that only a small amount of the energy striking upon the drum membrane enters that membrane. A large part is reflected back into the external auditory canal. It was also pointed out that the drum membrane yields but little of the energy it receives to the air of the tympanum. We are now prepared to consider the relation of the tension of the membrane in reference to the problems of sound transmission into the ossicular chain. The relation of drum tension to the amount of back reflection must be considered and the influence of drum tension upon the transmission of energy into the ossicular chain also taken into account.

It has been shown by Mangold and Eckstein[28] that "even if a true damping of sound vibrations may not be disputed and has been established during the voluntary contraction of the M. tensor tympani in E. (an individual), we must, however, emphasize that neither of us received the impression that an irritating sound stimulus is damped and rendered less irritating through the tension of the M. tensor tympani as determined by the noise of its own contraction. Even strong tensor contractions, which lead to distinctly recognizable tensor noises as well as to demonstrable pressure variations in the external auditory canal, show themselves powerless to damp the disagreeable sensation. The assumed protective reflex against strong and high pitch sound stimuli is even more problematic in individuals in which the contraction of the tensor in its assumed reflex function is not subjectively recognizable and, until this time, could scarcely be classed as objectively demonstrable."

The increase in drum tension, according to this statement, does not produce a pronounced damping effect. This must be interpreted according to the diaphragm-rod theory. The string telephone operates through a tense membrane and with its most tense area at the attachment of the transmitting element. If the medial displacement of the malleus increases the tension in the connective tissue of its periosteal attachment to the drum membrane, it will also increase the efficiency in transmission of sound vibrations from drum membrane to the malleus. Any damping effect therefore which may come about must be referred to the less efficient catchment through a more tense drum or to the subtraction of energy through the tensor tendon itself. Connective tissue under tension is a better transmitter than nontense connective tissue.

The point that the efficiency in the sound transmission apparatus depends in part on the positive pull of the malleus against the drum membrane is not merely a speculation. It lends itself well to the interpretation of the deafness caused by a hypertense drum membrane. Hypertension is usually due to the medial displacement of the drum membrane through air absorption or through adhesions. The connective tissue between the drum membrane and malleus is relaxed in these conditions and sound transmission from drum membrane to malleus impaired. It appears that the M. tensor tympani through its contraction will tend to damp out mass movements through the increased "weighting" of the drum membrane itself.

Both the Burnett and the Nagel and Samajloff experiments appeal to the writer as insufficient in the determination of the optimum, and in particular the minimum energies. Both series of experiments appear to show that mass oscillations of the drum membrane may arise under circumstances of unusual intensity in sound vibrations. One would not test the efficiency of a dictaphone by removing the mouthpiece some distance from the lips and whispering softly in its general direction. The two experiments noted in the previous paragraphs demonstrate the "damping out" effect of the external auditory canal and the drum membrane, even when the sound vibration is conveyed directly to the external canal. The damping effect will be more pronounced when the sound pulse undergoes a lateral dilution in its "side slip" at an angle into the external auditory opening. We must also bear in mind the surprisingly small amount of additional damping which even large masses of cerumen occasion. This will be discussed in Chapter III.

Kato has considered the problem of the "damping out" of the sound vibrations in their transmission through the ossicular chain, and the reader is referred to his article for details. "While it has been more or less generally accepted, in spite of the evidence to the contrary presented by Bockendahl, that the intrinsic muscles respond to sound vibrations with lightning-like contractions, comprehensive and repeated experiments have demonstrated that both of the intrinsic muscles respond with prolonged contractions as long as the sound may operate." "Opposed to this, however, are the findings that actual medial displacement of the ossicular chain does not occur as a result of the reflex contraction of the M. tensor tympani ,because the M. stapedius always responds and fixes the stapes. The M. tensor tympani reacts to stronger stimuli while the M. stapedius responds to weaker intensities; the contraction of the former is therefore always accompanied by reaction in the latter. However, no actual medial displacement of the stapes ever occurs, as has already been pointed out. This would only take place if the M. tensor tympani contracts independently, under which circumstances (with the M. stapedius out of commission) a medial displacement of the stapes may be observed in the reactions of the membrana tympani secundaria. Inasmuch as muscle contractions arising from sound vibrations never occasion an increase in the perilymphatic pressure, the 'damping out' effect suggested by Zimmermann (in terms of increase in the tension of the membrana basilaris) may not be considered."

"In conclusion, some mention must be made of the manner in which the intrinsic muscles perform their function of 'damping out' the intensity of sound vibrations. There are two possibilities: either the fixation of the stapes and the more marked drum tension results in a decrease in the amplitude of the vibrating masses, or a diminution of the intensity with which the sound vibrations are transmitted to the labyrinth is brought about through these changes. Naturally it is possible to conceive that both mechanisms may operate."

Kato's experiments on the reflex reactions to sound vibrations in the rabbit and the cat are extremely instructive. They give a possible test for acuity and range in hearing in various forms which is much more satisfactory than any hitherto known. The cat responds reflexly to pitches of 200 to 300 vibrations per sec., while the rabbit reacts to 128 and occasionally to 64. The fact that the M. stapedius contracts to 50,000 vibrations per second, which is entirely beyond the range of hearing in the human being, indicates a molecular transmission rather than a mass response. It is impossible to conceive a mass response in sound transmission apparatus to a frequency of 50,000.

The rabbit reacts to the pitch A3 at a distance of 60 m., but if the external cartilage is removed this distance must be cut down to 29 m. Removal of the drum membrane reduces the distance still farther to 8 m.; while reflex contractions are, however, again provoked with the pitch A4 at a distance of 5 cm., with malleus and incus gone and even with the stapes removed, reaction in the muscle may be observed. if the source is applied immediately to the external canal. This evidence is very important in considering the efficiency of the damping effect of the external canal, and also the efficiency of the membrane and ossicular chain route for the transmission of sound vibrations as opposed to the direct route through the medial tympanic wall or round window as suggested by Zimmermann and others.

The normal cat under certain test conditions becomes reflexly deaf in about five hours, while rabbit under similar circumstances is deaf in from five to six minutes. The time is markedly reduced if one of the muscles is out of commission, and still more markedly reduced if both muscles are cut or thrown out of function by means of profound narcosis. It has also been shown in various mammals and birds that cochlear degenerations occur earlier as a result of the exposure to prolonged high pitch vibrations in animals with the intrinsic muscles incapacitated than in those with muscles intact. Objections may be made to these experiments in that the animal is subjected to abnormal conditions of sound intensity. The extent of the lesions in both animals and birds indicates that at least two factors operate in producing the results. First, a fatigue and possible degeneration of a small cochlear area as a result of overstimulation; and, second, the spreading and relatively indefinite area of degeneration shows that mass movements in the perilymph are also probably responsible for the greater part of the resulting lesion. The relation of the problem of experimental cochlear degeneration and its bearing upon occupational deafness may not be taken up at this time.

It is, however, permissible to indicate that further work is demanded on the problem of the damping out of sound vibrations. Attention has already been called to the effect of the position of the external auditory canal and to the "side slip" of the sound wave front entering it. Investigations also appear to show that a great amount of the original energy entering the canal and registering in the form of drum membrane responses is "damped out" through reflection from the tortuous walls of the canal itself. Foley's sound pulse photographs indicate that a very large amount of the original energy striking upon a thin celloidin membrane is reflected back toward the source. Finally, the writer has emphasized the oblique position and the physical character of the drum membrane as objections to a mass response as a result of normal or optimum amplitudes of sound vibrations. It does not appear likely that the muscle tension or the stretch of the elastic ligaments can subtract any very material amount of the energy passing through the transmission apparatus. This appears to conform with the inability, voluntary or otherwise, to actually damp-down or damp-out sound vibrations. The damping response must be correlated, however, with the experiments of Kato, where he finds a stapedial contraction to extremely high pitches (50,000). He also obtained reactions to sounds of reasonable pitch at a distance of 60 m., which is probably beyond the range of the explosion or concussion waves arising from the sound source.

There can be little doubt that the rabbit hears the sound indicated in Kato's experiment at a considerably greater distance than 60 m. The fact that the response was not elicited beyond this distance with the external ear intact or at more than 29 m. with the external ear removed will show that sufficient energy is being applied to cause mass response of some kind and contraction of the muscle to "damp out" these movements of the stapes as a whole. This is to the writer a very important point because it indicates that the M. stapedius is definitely opposed to mass movements in the sound transmission system. The amount of molecular transmission is already limited through the size of the catchment area itself and by the factors already noted. It appears unlikely under normal conditions that sufficient molecular response through the drum membrane and ossicular chain could be produced to effect an actual degeneration of the cochlear apparatus. However, the excessive amplitudes could, and undoubtedly would, provoke mass responses which the intrinsic muscles might eliminate as long as they are functional. This is equivalent to saying that the cochlear degenerations arise at least for the most part from mass movements in the sound transmission apparatus due to excessive amplitudes. As long as the muscles are physiologically capable of cutting down these mass movements, the reflex deafness does not come about. It is not fair to assume that the rabbit has a keener sense of hearing than the cat and therefore becomes deaf in five minutes under conditions that produce reflex deafness in the cat in five hours. The cat may be able to damp out vibrations more readily.. It may also flatten out its external ear and offer a certain amount of additional damping out to the diffraction of sound vibrations into the external canal. This is apparently what takes place, because the cat not only flattens the ears against the head but also presents its tail toward the sound source.

The deafness which comes about through lateral displacement of the drum membrane, either due to a plus pressure in the middle ear or relaxation of the M. tensor tympani, or both, is probably not due to a disengagement of the incus from the stapes, which, as Bezold has noted, breaks the contiguity in the ossicular chain. It has been mentioned that the passive traction system of incudal ligaments opposes the contraction of the M. tensor tympani. It therefore contributes to a lateral drum membrane displacement on relaxation of that muscle. The lateral displacement of the long process of the incus from the stapes will readily come about because the ligamentum annulare is a very strong stapedial attachment when compared with the delicate elastic capsular ligament at the incudostapedial articulation. This disengagement may be observed in the guinea pig, where the malleus and incus are united by bony ankylosis and where the lateral displacement of the malleus necessarily carries the incus with it.

If Secchi's observations[36] are correct, then the middle ear is normally under a slight plus pressure. A tonus contraction of the M. tensor tympani might be assumed to maintain a contiguity at the incudostapedial articulation. The problem is not to be regarded as definitely settled. The suggestion may, however, be made that drum membrane tension is, after all, only an incidental result and is not the purpose of the contraction of the M. tensor tympani. This will at least conform with the relations as they are observed in birds where the M. tensor tympani function does not appear to be definitely related to the drum membrane tension.

Kato's experimental evidence on reflex contraction of the intrinsic muscles and the reactions which may be interpreted in terms of acuity in hearing demonstrate that the drum membrane and sound transmission system are the efficient means of transmitting sound vibrations. This is in direct opposition to the contentions of Zimmermann, Kleinschmidt,[27] Secchi,[35] Kretschmann,[25] and others, that the transmission takes place through the air of the tympanum, either through the medial wall of the tympanum or through the fenestra cochleæ. The reader is referred to the excellent reviews of Treitel,[35] of Frütiger[17] and of Lucæ.[26]

The writer feels justified in presenting the diaphragm-rod theory of sound transmission and declaring that the mass movements ordinarily described lie beyond the physiologic maximum. In other words, the entire apparatus from drum membrane to hair cells is set in for molecular vibration. The moment mass movements of the drum membrane arise they are damped out in so far as this is possible. The sound transmission system therefore represents a most efficient area of bone transmission to the inner ear, and in particular is this true in small amplitudes (intensities) and long wave lengths (low pitch).

The registration of vibrations in the auditory cells may therefore be effected by the sound pulse passing on them or through them. Neither membrana basilaris nor membrana tectoria can effect the mechanical stimulus essential to a reaction. The fact that the auditory cells are hair cells and the fact that they are definitely related to a membrana tectoria may be due to the ancestral type of pressure organ or displacement organ from which they are derived. The membrana tectoria may perhaps offer inhibition to mass movements. It may be a sort of stabilizer. This X quantity, which occasions a reaction in auditory cells to vibrational frequency and intensity, is not particularly different from the behavior of the retinal cells. Is it really essential to our conception of the behavior of the light in the various media of the eye that the workings of the rods and cones be established? Perhaps then we have arrived at a point where our limitations confront us. We may assure a sensitization of cells to picking up frequencies and their amplitudes. We may harmonize this behavior with the extensive researches in cochlear degenerations as a result of prolonged auditory irritation. But we may also at this time remember the words of the immortal Goethe: "Eben wo Begriffe fehlen da stellt ein Wórt zur rechten Zeit sich ein."

It appears therefore that the drum membrane and entire sound transmission system is set in for appreciation of sound vibrations of small amplitudes. When amplitudes ot sufficient intensity are produced, the tiny mass vibrations of the drum membrane are absorbed and dissipated in the hinge system of the sound transmission apparatus and further inhibited by the contraction of the intrinsic ear musculature. The very minimum which the displacement theorists grant may therefore represent a maximum of hearing or even beyond the capacity of appreciation in terms of intensity of hearing.

The writer appreciates this last statement is a decided shock to anyone who has followed the mass displacement theories in any one of the various forms. Perhaps the first thought which comes to mind is this: if a balanced drum, a straight rod and an insulated opening in the otic capsule to the perilymph fulfill the requirements for sound transmission from air to end organ, why do we find a more elaborate mechanism with muscle attachments and elastic ligaments? Why is it essentialy that a fenestra cochleæ with its limiting elastic membrana tympani secundaria be found in practically all forms of terrestrial vertebrates? These questions, proposed by Beyer, may be quite successfully answered. If the explanation does not hold on the basis of comparative anatomy and physiology, then the diaphragm-rod theory cannot be employed as a working basis. If this be accomplished, the problem stands open for the tests of repeated experiment before the theory may be accepted. The essential differences between all displacement theories and that of molecular transmission consists in this—not only must the inner ear be physiologically guarded against any factors of displacement arising from sound vibrations, but all displacement factors arising in the middle ear must also be ruled out in so far as this is possible.

X. What is the function of the middle ear complex?

Keith remarks in relation to the Wrightson theory that we must seek an explanation in the comparative anatomy and physiology for matters which are not clearly defined in the human being. Objections were made to the confirmatory evidence proposed by Keith because the close agreement in the general structural pattern of the inner ear merely shows that the structure of a given part must bear some definite relation to the ancestry of the organ. This is neither a discovery of Keith's in reference to the inner ear nor does it substantiate the Wrightson hypothesis more than it does any one other of a dozen theories. It was also pointed out that the mechanical relations in the bird are less readily associated with the Wrightson hypothesis than they are in the mammal. This also obtains for any other displacement theory. The very similarity in structure will make any theory of hearing which includes the facts obtain for mammal and bird alike. In other words, if we solve the problem for one form we solve it for all forms within certain limits which are dependent on individual variations.

Curiously enough, the entire Wrightson hypothesis rests on a middle ear behavior; the mass reactions in the drum membrane; the "intensifying press" function of the ossicular chain; the muscle balance; the displacement of the stapedial footplate in relation to the membrana tympani secundaria. Yet Keith furnishes no evidence wherein the comparative anatomy such as that of another acutely sensitive form, the bird, furnishes a confirmation to the correctness of these fundamental conceptions. Perhaps therefore a study of the comparative anatomy and physiology of the frog, the bird and the mammal may furnish a clue to the complex middle ear system. It must be remembered that the middle ear region has a far more interesting phylogeny than that of the inner ear. It was stated in the beginning of this article that a consideration of the reptiles would be eliminated. This family presents an extremely variable picture which must, of course, not be disregarded. The reader is entitled to first hand information in so far as this is possible. We have not investigated the reptilian ear. Three forms have been selected—the frog, the bird and the mammal, because they represent three distinct possibilities in middle ear mechanics. The information on the frog has been largely derived through courtesy of Terence Ahearn, S. J.,[1] who is at present writing his doctor's thesis on the problem, undertaken in this laboratory. The reader is referred to Ahearn's article when it appears for a detailed description. The work on the bird and the mammal are a result of personal investigation. The report on the elastic ligaments of the cat (dog may also be included) are presented for the first time.

A. The middle ear mechanics in the frog. (Fig. 7.)

The drum membrane in the frog is relatively thick (d. m.) and is supported by an annulus (an.) of cartilage. The sound transmission system is roughly in the form of a twisted U, with one extremity of the U markedly thickened and the connection between the two arms is swollen. The cartilage portion (lateral) displays two definitely weakened areas at which bending occurs upon light pressures. Following the apparatus from the otic capsule (o. c.), the small descending process displays a hinge at (a) and then swells to be attached to the medial drum membrane surface. This attachment area runs upward, loses its connection to the drum membrane and forms a small process which bends sharply on itself at hinge (b) to continue medialward in an enlarged club shaped mass —the columella proper—which occupies the upper area of the fenestra vestibuli. It will be noted that the surface of this process is in relation to the perilymph (pl.) and is insulated by elastic ligaments from direct contiguity with the otic capsule at its upper surface. Below it is mortised into another cartilage element the operculum (o). It is joined to the operculum by elastic ligaments. The operculum is confluent with the otic capsule at another cartilage hinge (c) so that all movements of the sound transmission system are accompanied by accompanying movements in the operculum. The outward displacement of the cartilage is resisted by a stout elastic ligament (not indicated), while the inward displacement is resisted by the M. opercularis, the action of which is indicated by the arrow (m).

The inner ear system is represented as a hole in the otic capsule filled with perilymph (pl.) and containing a ductus cochlearis (stippled). The capsule has three chief openings—

the fenestra vestibuli filled by columella, operculum and elastic ligaments. The second opening is the foramen perilymph sup. (F.P.S.), where the perilymph liquid is separated from that of the spatium subdurale (S.S.) by a thin membrane. The third opening or foramen perilymph, inf. (F.P.I.), is closed in by an elastic membrane, the membrana tympani secundaria (M.T.S.), which faces the mouth cavity but is separated from the mucous membrane by a lymph sac (L.S.) and by muscle fibers (not indicated). It will be seen that the tuba auditiva (t.a.) is a short wide open duct leading from the mouth to the cavity of the middle ear.

When the frog swallows the air, the middle ear cavity is placed under a plus pressure with a resulting lateral drum membrane excursion indicated by the arrow +. This force, because of hinge "a," is translated at "b" into a reverse motion indicated by the arrow. The motion of the drum membrane outward tends to thrust the sound transmitting system into the perilymphatic space so that increases in pressure would escape along the lines indicated by the fine arrow—through the cochlear duct or around the cochlear duct (helicotrema) toward the foramen superior and foramen inferior. Minimum motions are probably compensated for in the vascular supply. However, larger movements are prevented by the contraction of the M. opercularis. This muscle is therefore opposed to both displacement factors in the columella proper. It is also interesting to see that if a medial displacement factor is applied to the drum membrane (arrow x), the area at hinge 'b' will become applied to the drum membrane and the resulting motion in the columella will be the same as before.

The mechanism in the middle ear in the frog is a response to an adjustibility in the sound transmission system which arises because of the lateral drum membrane motion. This motion is dependent on respiration and allows a maximum excursion at the drum membrane area with a minimum response at the perilymphatic end. The active phase opposed is the plus pressure due to air injection. It may also be stated that according to the diaphragm-rod theory an inefficiency in the apparatus may be seen in the direct connection of the sound transmission system to the otic capsule, and in the thickness in the membrana tympani. The articulation of the columella with the opercular cartilage merely indicates a large adjustment area toward preventing pressure fluctuations in the perilymphatic system. It is also necessary to bear in mind that the helicotrema in the frog is an extremely wide opening offering none of the objections of resistance to displacement mentioned by Wrightson for the mammal and bird.

While the frog does hear certain things, its sound transmission system and drum membrane do not appear particularly well adapted. Nor does the development of the inner ear suggest a refined sense of hearing.

B. The mechanics of the bird's ear. (Fig. 8.)

The schematic diagram of the bird's inner ear shows similarity to that of the frog excepting that the membrana tympani secundaria (M.T.S.) (fenestra cochleæ) has rotated to face the middle ear. It must be remembered in some birds (the goose, etc.), the fenestra cochleæ is separated from the cavum tympani by the vena jugularis. The membrana tympani (D.M) in birds is thin and markedly convexed outward into the external auditory canal; the two middle ear cavities are confluent, and the two tubæ audivitæ (t.a.) open through a common tubal duct (t.d.) into the mouth. This small opening is normally closed. The sound transmission system consists of a cartilage tripod which occupies the posterior drum area. The M. tensor tympani attaches to one element of this tripod. This muscle is extratympanic and the line of its pull is indicated by the arrow M. The muscle is resisted by elastic ligaments, notably the columellar-squamosal ligament (c.l.) of Platner. The cartilage apparatus joins the bony columella just medial to the attachment of this ligament. The bony columella (C) is attached to the margins of the fenestra vestibuli by elastic ligaments, and the membrana tympani secundaria is also elastic in structure (M.T.S.). The otic capsule is now well separated from the cavum subdurale, and a hole is left in it to represent schematically the aqueductus cochleæ and aqueductus vestibuli (aq.). It will also be noted that the membrana vestibularis (Reissner's membrane) is indicated in a heavy vascular tegmentum vasculosum (T.V.).

When the tubæ auditivæ open freely to the mouth, the air pressure on each side of the drum membrane is probably equal. Displacement forces arise with closed tuba, either through air absorption or through positive pressure at the external auditory canal. The latter tends to thrust the columella inward and beget increased pressure in the perilymphatic spaces. This increased pressure will be absorbed by the blood vessels (note the position of the tegmentum vasculosum), or may escape through the aqueductus cochleæ et vestibuli or at the membrana tympani secundaria. The contraction of the M. tensor tympani directly opposes this and tends to hold off the inward drum membrane displacements from increasing the pressure in the perilymph (pl.). Negative pressures in the external canal are also resisted by the elastic ligaments and compensated by the great mobility in the drum membrane. There are also a series of cartilage hinges in the sound transmission system which allow a more pronounced reaction in the tripod cartilage than in the bony columella proper (see writer's article, American Journal of Morphology).

The mechanism in the bird's ear is one set in for variations in drum membrane position. The compensation system is found in an adjustibility of the sound transmission apparatus and is of direct physiologic importance. The muscle tensor tympani does not figure conspicuously in terms of drum tension, although this necessarily results. Rather the M. tensor tympanum in birds combats active displacement factors which tend to produce excesses of perilymphatic pressure. The cochlear area in birds is poorly placed to be acted upon by sound vibrations direct through the otic capsule. The range of adjustibility of blood vessels in the perilymph system is even better developed than in the mammals. It may also be mentioned that the columellar footplate does not lie at the end of the cochlear duct but faces its middle area.

C. The mammal. (Fig. 9.)

The schematic diagram presents the relations in the human being, and some reservations must be made in carrying over this scheme to the cat, the guinea pig and in particular the mouse. The two middle ear complexes are completely separated. A new cartilaginous tuba auditiva is shown as closed with the function of the M. tensor veli palatini (M. dilator tubæ) to open it indicated by the arrow M. d.t. The drum membrane is drawn in and attached to the malleus. The position of the incus and stapes is indicated. The pull of the

M. tensor tympani is represented by the arrow at M. t., while that of the M. stapedius is shown by the arrow M. s. The inner ear is about the same as that pictured for the bird and frog, which will indicate the purely schematic presentation. The heavy vascular tegmentum vasculosum in the bird is replaced by the delicate nonvascular membrana vestibularis.

When the tuba auditiva is opened, the M. tensor tympani appears to relax, allowing lateral displacement of the drum membrane. This lateral displacement of the drum membrane may be due to several factors: first, the intrinsic elastic structure of the drum membrane, which will tend to decrease the umbo-depth slightly; second, the incudal elastic ligaments and character of the incudomalleolar articulation may contribute to a positive push factor outward of the malleus; third, the manner of tubal closure may create a positive pressure within the middle ear region as Secchi has suggested; and lastly, a minus pressure in the external auditory canal (barometric) will tend to draw the drum membrane outward. It appears that the drum membrane at rest is not placed in a position of physiologic efficiency and tendency for a disengagement at the incudostapedial articulation must be counteracted by muscle pull. This mechanical requirement in the mammal does not obtain in any other vertebrate. The diagram presents a possible interrelation between the M. tensor tympani and the M. tensor veli palatini. The arrows indicate the former muscle is set in against any factors which will make for a lateral drum membrane displacement. The medial displacement of the ossicular chain tends under unusual conditions of pressure to force the stapes toward the perilymph and beget increases in perilymphatic pressure. This is opposed through contraction of the M. stapedius. The distribution of the possible excess pressure in the perilymph, apart from that taken care of in the minute veins, is shown in the arrows within the inner ear.

We must grant, on the basis of Kato's experiments, that reflex contractions of the M. tensor tympani do not give rise to fluctuations at the membrana tympani secundaria. It must also be granted that the M. stapedius absolutely opposes minute displacements of the stapedial footplate. The vascular regulator mechanism compensates for what would constitute mass movements in the sound transmission system in reference to any motion created by responses of the drum membrane to sound vibrations of usual intensities. The compensation area in the membrana tympani secundaria will therefore not be called for in relation to either sound vibrations or to reflex contractions of the M. tensor tympani. It will only be effective in the larger drum membrane fluctuations which in the mammal are necessarily limited. It has been mentioned that the relatively larger fenestra cochleæ in birds is suggestive of a compensation area for perilymphatic adjustments to mass movements in the sound transmission system. These movements are not a result of sound vibrations but are due to barometric fluctuations and to air absorption.

The double displacement factor in mammals therefore calls for two muscles. A new muscle, M. tensor tympani, is related to factors which tend to produce a lateral displacement of the drum membrane and probably maintains a contiguity in the sound transmission system at the incudostapedial articulation. The second muscle is similar in function to the single M. tensor tympani in birds or the M. opercularis in frogs. The M. stapedius affords protection against increases in pressure through the application of a movable skeletal element to the perilymphatic space. Both of these muscles tend to inhabit mass movements in the sound transmission system.

Throughout the entire comparative anatomy of the middle ear region three prominent features appear: first, a veriability in the position of the drum membrane not dependent on hearing but on displacement factors arising within the middle ear; second, an adjustibility in the sound transmission system gearing down the mass drum membrane movements in relation to the fenestra vestibuli. These are not related to sound vibrations in their propagation to the perilymph but merely decrease the range of mass motion. Third, protection is afforded to the delicate end organ through muscle contraction against increases in pressure brought about through drum membrane displacements and in inhibition to the mass responses propagated from the drum membrane toward the perilymph. This appears to harmonize more completely with the comparative anatomy and physiology of the middle ear region than any suggestion thus far published. The work on the intrinsic muscles of the mammal by Kato and the investigations on the human M. tensor tympani by Mangold are certain to stimulate researches along these lines. It is also important that the tubal mechanics and the function of the M. tensor veli palatini be included.

It is only fair to state that the contractions of the M. stapedius to the sound vibrations, as noted in the experiments of Kato, are not readily interpreted. We must assume that the M. stapedius is always in a state of tonus contraction. When a sound is superposed upon the sound picture of the environment to the point that it begins to occasion a mass reaction or a tremble in the stapes, an additional muscle contraction is manifested. This statement means that the stapes, because it is extremely light and because the energy is discharged from the footplate into a medium of shorter wave lengths, may have sufficient restoration forces connected with it to undergo mass reactions. The effect of the M. stapedius in its contraction, as confirmed on experimental animals, would be to act as a damper.

The comparative anatomy and physiology affords certain evidence which distinctly favors the diaphragm-rod conception of the sound transmission. It may be well to reserve judgment on whether this, that or the other theory is the correct one. It is our contention, however, that the older view of a molecular transmission has certain things in its favor. It should at least be included in our discussions of the problem. In particular, the diaphragm-rod theory lends itself well toward the solution of several features in deafness which have been considered as hopeless. Perhaps they have been considered hopeless because the mechanics of sound transmission have been looked upon as quite well established by Helmholtz.

We may take the liberty, in closing this chapter, to refer again to the opening paragraphs in this article, where we call attention to the fact that the "functional adaptation" and "structural adaptation," after all, are merely terms which indicate that we have approached the region where cause and effect may not easily be separated. It may come to pass that what we have been considering as the active stimulating factor in sound analysis—the membrana tectoria, membrana basilaris and the delicately refined histologic structure of the end organ may, after all, be nothing more than an earmark of the ancestral pressure or displacement organ from which it was developed. The resultant microscopic picture must be followed with this point in view rather than believing it will necessarily explain a selective function in the auditory cells. Let us remember that whatever the method is by which these cells operate, even under the lowest tones, the ear is quicker in its response than the eye. The discrimination of a suppressed vibration up to a frequency in high pitches which exceed that of touch (130 per sec.), implies that marked after-images such as found in the retina do not occur. This is an important point to bear in mind, because a sympathetic vibration does not omit vibrations when the vibrations are eliminated from the activating source. The inner ear therefore appears to operate under no conceivable conditions of inertia, which indicates the activating agent as a sound pulse and not a mass response in the sound transmission system.

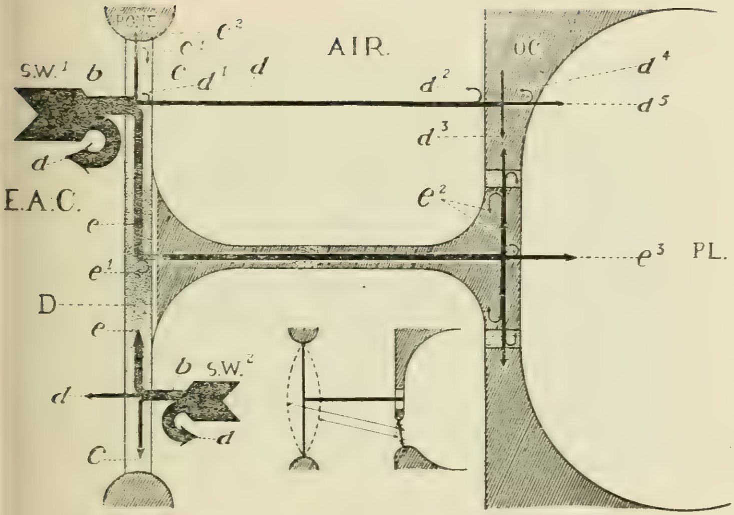POLE
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Fig. 2.

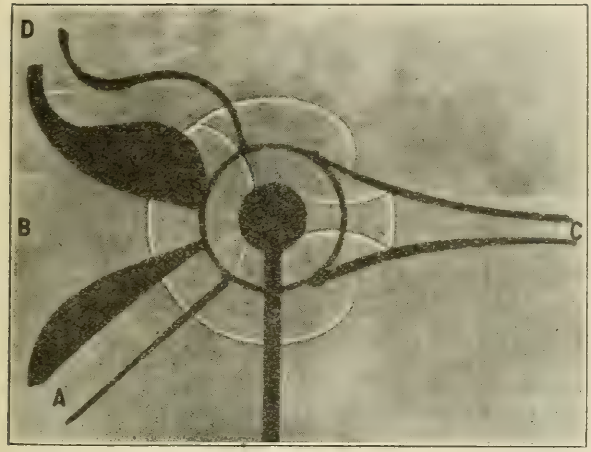


Fig. 3.

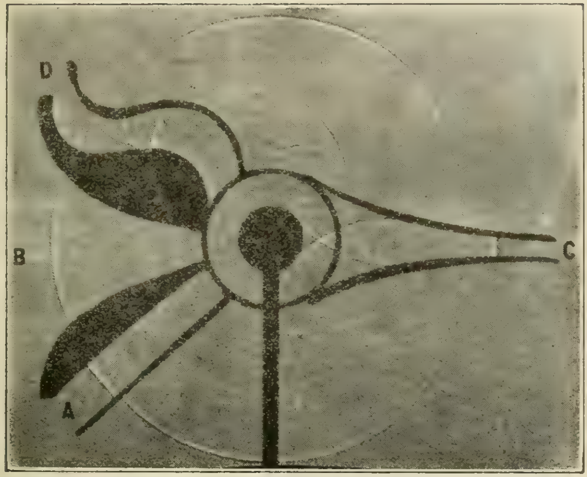
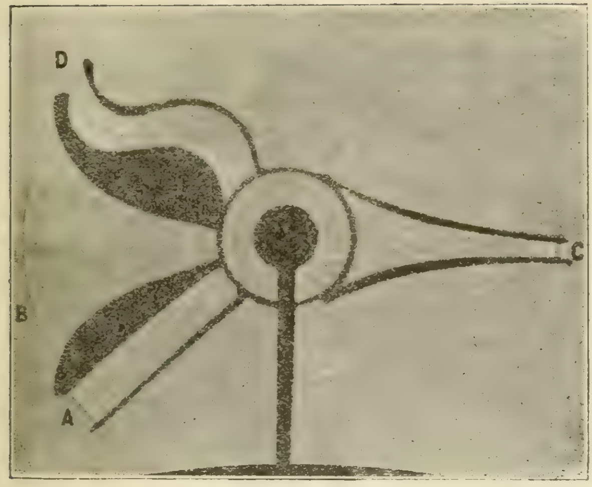


Fig. 4.



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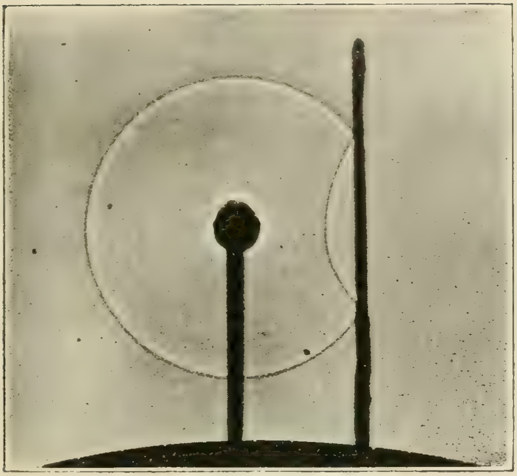

Fig. 6.

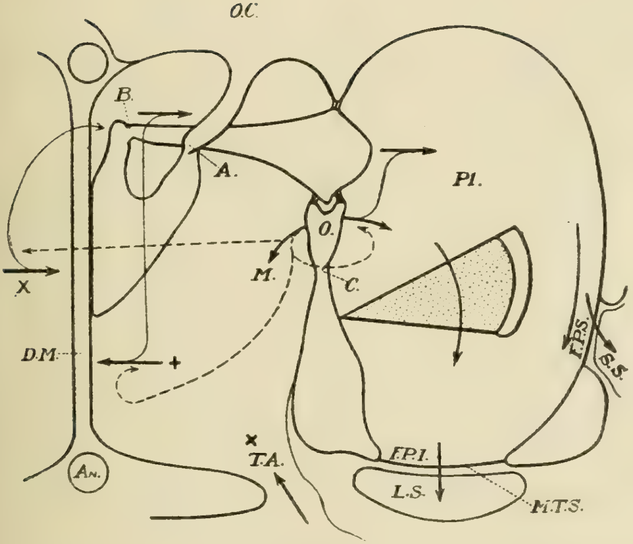

Fig. 7.

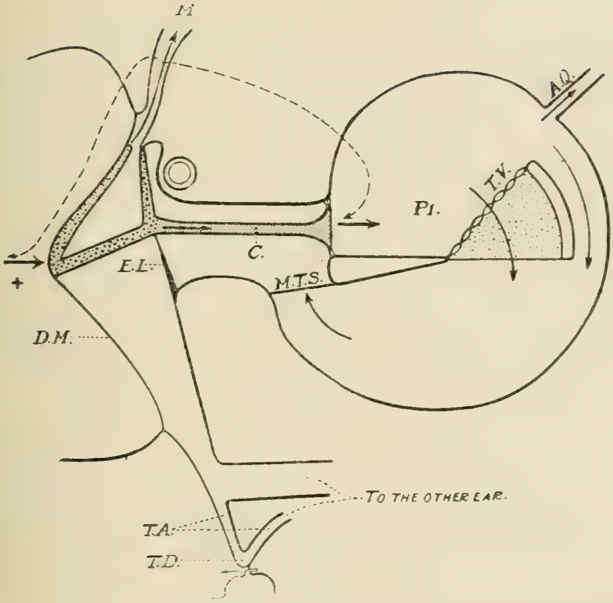

Fig. 8.

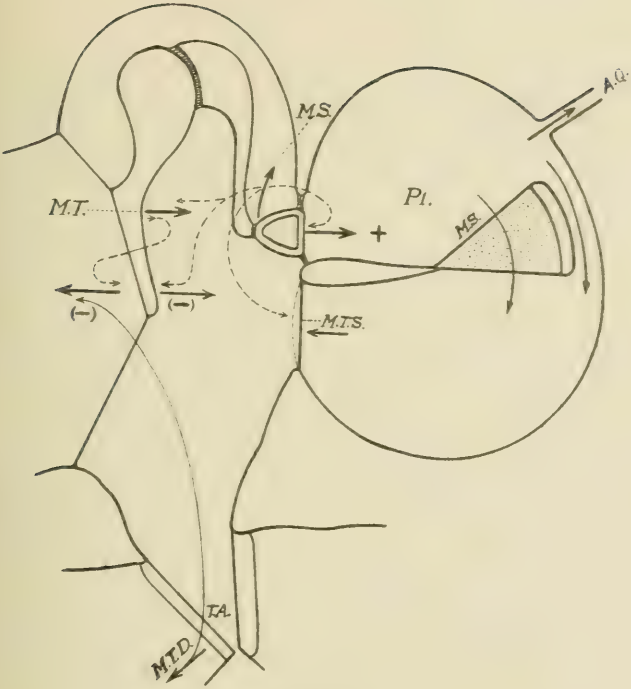

Fig. 9.

XXIX.

MISCONCEPTIONS REGARDING THE IMPORT OF RETRACTION OF THE DRUM MEMBRANE.

By Edmund Prince Fowler, M. D.,

New York City.

Elemental physiology of the ear teaches that normally the drum membrane is made convex peripherally and concave or funnel shaped centrally, by its attachment to the more mesially situated manubrium, and that the air pressures upon its two sides are approximately equal owing to free communication with the surrounding air, constant by way of the external meatus on the one side, and periodic by way of the eustachian tube upon the other.

The drum membrane is then normally retracted, its central fibres are tensified and curved from the plane of the annulus toward the manubrium, and markedly toward the umbo.

Some confusion or carelessness exists as to the terminology of abnormal drum tensions, hyper and hypotension being used erroneously to denote respectively lack of or increased mobility of the drum. As a matter of fact, hypertension denotes immobility only in the sense of an increase in strain upon the drum; whereas lack of mobility may or may not accompany such a strain. Likewise hypotension means a loss of tone or lowered strain upon the drum fibres so that increased mobility will coincide with such loss of tone only if there are not limiting adhesions, etc.

We use the term retraction to indicate an abnormal sinking in of the tympanic membrane, and as this may be due to a relatively lessened air pressure upon its inner side, it has become an almost universal habit to think of the condition as diagnostic of negative middle ear air pressure, and of the relief experienced from inflations as due to the restoration of more equal pressures upon the two sides of the drumhead.

However, when we examine retracted drums, it is amazing to find that in but comparatively few instances can any negative air pressure in the tympanum be demonstrated. Even in marked cases, with great foreshortenings of the manubrium and prominence of the processus brevis and posterior fold, there may be no vacuum within the middle ear.*

How, then, are we to interpret retraction of the drum? I believe we may better understand the matter if we will give it a little thought along the lines I am about to indicate.

The walls of the eustachian tube, being soft and practically in contact for a considerable distance, are easily approximated more firmly by inflammatory or other swellings in the mucosa or submucous connective tissues. When such occlusions of of the tube endure for some time, we are led to believe that there ensues sufficient air absorption from the middle ear and its connecting spaces to appreciably retract the drum. This inward negative pressure tensification may cause diminished hearing, often tinnitis, and at times vertiginous sensations. It also sucks together the walls of the tube (in like manner to the inspiratory effect upon the gill valve of a gas mask) and thus makes more permanent and firm the tubal closure.

If instead of or beside tissue swelling there is present an exudate in the tube, this fluid will not only tend to close the tube as such, but on account of its viscosity will cause the tubal walls to adhere, or as the direction of the cilia motion is pharynxwise and all transit through the tube can be demonstrated to be more easy in this direction (and for this reason) there will be instituted another cause for exhaustion of the tympanic air, namely, a suction brought about by the downward movement of the fluid. Depending upon the position of the head, gravity may aid or hinder this movement. I have been able to demonstrate only during deglutition and automatic movement of fluid from the middle ear into the tube.

Any lessening of pressure in the tympanum will cause the external atmospheric pressure to predominate, and the drum with its movable attachments will be forced inward. Two main factors, then, may operate to cause retraction of the drum after occlusion of the tube: First, absorption of middle ear air; second, movement of the fluid contents of the tube toward the pharynx, mainly by cilia motion, and exhaustion of the middle ear air by such hydraulic suction.

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*The method for determining air pressures behind the drum was described in detail last year before the American Laryngological, Rhinological and Otological Society and the Otologic Section of the A. M. A.

If the negative pressure is of sufficient strength, a fluid exudate is expected to fill the tympanum to a greater or less degree.

After failing to find any relative data in the modern or ancient literature, an attempt was made to determine the exact amount of negative pressure requisite to cause an exudate from mucous membranes. Small cupping glasses applied to the buccal mucous membrane gave no definite findings; in fact, different persons appear to be affected in varying degrees by like pressures. The Schneiderian membrane also gives various results under similar negative pressure in different persons. In hypertrophic rhinitis, an exudate is obtained after a short period of suction; in atrophic rhinitis but little effect can be produced even with strong suction (over 40 or 50 mm. Hg.) applied for long periods of time (one-half to one hour). This, of course, would be logically expected, and I believe similar lesions within the middle ear and tube may account for the ready, or tardy, or non-occurrence of serous or mucoserous exudates accompanying retraction of the drum membrane. In like manner, hypertrophic and atrophic otitis media must influence the ready, tardy, or nonoccurrence of air absorption.

Small negative pressures, then, do not cause an exudate into the middle ear; but from the observation of many cases I feel certain that only small negative pressures (5 to 20 mm. Hg. at most) accompany otitis, and that if there be caused marked retraction of the drum this cannot be due to their strength, but rather to the prolonged or oft-remittent presence of negative pressure, first weakening and then allowing of marked recession of the drum.

Positive external meatal or negative middle ear pressures up to 20 mm. Hg. depress the drum membrane but little compared to pathological retraction.

I have examined scores of cases with apparently a complete tubal closure, wherein no negative air pressure within the middle ear could be demonstrated by any of the methods I have described for checking this factor. There can be but one explanation for such a failure, namely, no appreciable air absorption is occurring in these cases. No other deduction seems tenable. Such cases also frequently show little retraction of the drum. Here again there is present no negative air pressure. It is obvious that when the tube is functioning normally, no negative pressure will long endure within the middle ear spaces, and no air absorption operate to retract the drum; but with open tubes marked permanent retractions of the drum are common.

There is some doubt as to just what constitutes a patent tube. I would say that a physiologically patent tube is one which will permit automatic ventilation of the middle ear, especially during swallowing, whether the air in the tympanum is of normal pressure or of lessened pressure due to prior deflation from any cause.

Another finding is that after firmly plugging the external meatus with various cereates, etc., no absorption of air from the external meatus may be demonstrable, perhaps even after days of waiting.

By showing how slow may be the air absorption from the body spaces, it is only necessary to remind you that it takes often 8 to 12 days to absorb 1,000 to 2,000 cc. from the peritoneal cavity, after inflations incident to X-ray diagnosis.

We may thus account for the failure of meatal plugs to effectively hold or pull the drum outward through air absorption. Has any one ever examined a drum after subjection to the supposed suction from meatal plugs and found it other than in its previous retracted position? I think not. This failure occurs even when the tube is patent. Again, no appreciable air absorption has occurred from the tissues. The external meatus epithelium may be considered to approximate roughly an atrophic state of the mucous membrane of the middle ear. In such states it is reasonable to doubt the ready absorption of air.

Some one will say: Well, you will admit that the drum cannot be retracted without something holding it back. Yes, but, in a way, it may be. How, then?

For the same reason that a bowl is hollow, a section of pneumatic tire concave, a soft hat crinkly. There is insufficient tension at the periphery to maintain the fabric as a flat surface, and the redundant material must take a position to one side or the other of the peripheral attachments. Why, then, is the concavity of the drum usually outward, if there is no difference between the air pressures upon its two sides? Because: First, during the establishment of the retraction, portions of the drum become set in this position and so remain, unless forced outward by inflation or external meatal suction. In fact, slight increases in middle ear pressure may fail to cause local retracted portions of the drum membrane to bulge outward, and strong inflations to maintain bulging but for a few minutes, far too short a time to account for their collapse by air absorption on their mesial side; second, the tube, not functioning physiologically, there is a tendency to maintain in the middle ear a stationary air pressure. This tendency is often observed, not as an active force in increasing retraction but simply as a preventive of restoration to the centered position of a weakened drum. If there were present a measurable negative pressure, constant stretching of the drum and a constant increase in the retraction would occur; but that such need not be so is evident from the many cases which apparently remain for long periods in status quo. Third, adhesions or fibrous bands between any part of the middle ear and the retracted portion. These may be weak or elastic enough to admit of quite free movement of the membrane, and yet sufficient to cause its return to the retracted position upon removal of the replacing force. They may be difficult to demonstrate.

Of other causes for retraction, I will not speak at length at this time but, needless to say, of the utmost importance are adhesive processes, displacement of the ossicles, shortening of the tensor tendon, subluxations, stretching of the ligaments, etc. All these have, I believe, more to do with permanent retraction of the drum membrane than has air absorption.

CONCLUSIONS.

Hypertension is not synonymous with immobility of the drum membrane. Hypotension is not synonymous with increased mobility.

Air absorption may be the primary cause of retraction of the drum membrane. It is never over a few millimeters (under 20) of negative pressure, greater in hypertrophic or congested states: practically nil in atrophic states, and in the latter seldom if ever the primary cause of exudate into the middle ear. It cannot operate with patent tubes or with perforations. It often is absent with closed tubes. It is usually unmeasurable except in active stages of tubal or middle ear congestion. The lesions accompanying retraction of the drum are of more importance from a clinical standpoint than is the negative pressure.

Permanent retraction of the drum is seldom if ever due to the pressure of negative or diminished air pressure within the middle ear. This factor as an accompaniment or cause of drum retraction has been and still is greatly exaggerated.

114 East Fifty-Fourth Street.

XXX.

ACUTE INFECTIONS INTO THE SEBACEOUS GLANDS AND HAIR FOLLICLES OF THE NASAL VESTIBULE.*

By Lee M. Hurd, M. D.,

New York.

The common and usually trivial boil in the nose may suddenly terminate fatally by extension of the infection through the venous channels to the cavernous sinus. This is not realized by the laity and is not, I fear, any too well recognized by many physicians. When only two text books on rhinology mention furuncles and do not hint, even, that there might be grave complications, and also, when only a few text books of general surgery mention the subject, it is no wonder that this condition is considered so lightly. It is a striking fact brought out by the literature that most of the cases are only recognized when they are well advanced and hopeless, usually by the consultant who reports the case.

If anything is to be done to cut down this mortality it will be by early recognition that the cavernous sinus is threatened and by very prompt measures taken to present it.

Etiology.—Predisposing and exciting causes are similar to those of furuncles elsewhere on the skin, with the added bad habits of picking nose and pulling out the vibrissae. The staphylococcus is the common organism but it may be streptococcus.

Symptoms.—Pain, tenderness, redness and circumscribed swelling in the vestibule sometimes extending through to the skin of the external nose. Later softening at centre with rupture occurs and discharge of pus and slough "core" followed by prompt healing.

This trivial discomfort may suddenly become extremely serious by extension to the cavernous sinus, due either to attempts at opening, usually with a dirty needle at home, or, by squeezing, rupturing nature's barrier, driving the infection into the less resistant subcutaneous tissue, or picking the head off the furuncle with a dirty finger nail, or, by too free incision, opening up new avenues outside nature's barrier. In other cases it seems to travel through the venous channels by reason of lack of resistance to infection.

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*Read before the American Academy of Ophthalmology and Otolarygology, 26th Annual Meeting, Philadelphia, Pa., October 17, 1921.

Diagnosis.—A furuncle, or collection of them, should offer no difficulty in diagnosis; in fact, the patient usually has correctly diagnosed his red, swollen, painful nose as a boil and has usually operated with a needle and squeezed it, seeking medical aid because it is getting worse under his treatment.

Complications.—There may be sloughing of the inferior lateral cartilages.

The cavernous sinus becomes infected more frequently than is generally thought and it is one's duty to forestall this calamity, if possible, for the mortality of cavernous sinus thrombosis is practically 100 per cent.

It behooves us to manage the case so that the infection will not reach the sinus, if it can be avoided.

According to the literature, one feature of nearly all the reports is that the cases were not seen until too late and diagnosis made still later. Also, that the vestibular furunculosis is not well classified, cases being in with other case reports. As to when and how the primary focus was treated is not clear enough to draw any constructive deductions, whether the cavernous sinus thrombosis came from either neglect or mismanagement, or was so fulminating that nothing could be done.

With a vestibular infection a tentative diagnosis of cavernous sinus thrombosis should be made on the appearance of any of the following eye or orbital signs which should be considered very suspicious: Swelling of the lids, chemosis, pupillary changes, deep seated headache, beginning ptosis and exophthalmos, lacrimation, congested retinal veins, symptoms of sepsis. Later, when ophthalmoplegia, pronounced chemosis, edema of the lids and exophthalmos plus cerebral and septic symptoms develop, the case is hopeless and it usually is only a matter of a few hours before the opposite eye indicates that both cavernous sinuses are thrombosed and dissolution is near.

Treatment.—Though most of the vestibular furuncles are trivial affairs, it should ever be remembered that, from poor resistance of the patient or bad management, a seemingly trivial affair may explode with fatal results.

When fortunate to be seen early, my procedure is to use wet dressings inside and out and, as soon as there is evidence of pointing, to make a small incision at this place and never to go outside of nature's barrier, and by applying suction to draw out the infective material, fill the cavity with bismuth paste between treatments, and never to squeeze the swelling which might cause rupture into the subcuticular tissue where the chances of tissue resistance are much lower. This treatment seems to be much better than extensive incision and expression which is so often mentioned in the literature of the cases which later developed cavernous sinus thrombosis. I have treated a number of cases in which the local involvement was severe, yet with a small incision entirely within the barrier and with suction, they have recovered without complication except sloughing of inferior lateral cartilage in three cases.

As this infection extends along the anterior facial, angular and superior ophthalmic veins to the cavernous sinus above and perhaps by the deep facial vein and pterygoid plexus below, it seems rational to ligate the anterior facial vein near the inner canthus, as Bullock[1] reports in one successful case of upper lip infection, and also to ligate the anterior facial above the deep facial vein at the anterior border of the masseter muscle as soon as the infective area is increasing instead of diminishing or, if there is the slightest orbital or ocular symptom of cavernous involvement.

As lateral sinus thrombosis sometimes recovers, similarly, why may not the cavernous sinus overcome a slight infection if further supply is stopped? At least facial vein ligation is a simple procedure and may save the patient's life.

As surgical attack of the cavernous sinus for anterior infections has up to the present failed to cure ([3 4 5 6 7 8]) it has in a few instances even failed to open the sinus, but further technical experience and earlier diagnosis will probably produce better results from the use of one of the different methods, in what is now a hopeless situation.

39 East 50th Street.

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XXXI.

"VARIATIONS OF INTRACRANIAL SYMPTOMS FROM FRONTAL SINUSITIS."*

By Louis Levy, M. D., F. A. C. S.,

Memphis, Tenn.

In preparing this paper I have tried in the following pages to bring out the various intracranial symptoms that are presented during or following attacks of frontal sinusitis.

Realizing from experience that in no two patients are these symptoms the same, it can readily be understood why no set rules can be applied in making a diagnosis of extradural or intradural complications.

It is natural that the malignant tendency of sinusitis may manifest itself by its extension through the cerebral wall of the frontal sinus and result in the meningeal symptom of frontal lobe abscess. It is therefore well, when these symptoms appear, to be on guard for the possibilities of cerebral complications.

In reviewing the literature regarding the intracranial complications following frontal sinusitis, it is surprising that such a small amount of attention has been given to the subject. This may be due to the fact that abscesses in the frontal lobes cause no early symptoms and paralysis takes place only when the involvement becomes generalized.

In 1914, Boenninghaus reported eighty-seven abscesses of the frontal lobe, caused by frontal sinusitis. In 1919, Gerber made a careful review of the literature and reported in all two hundred and forty cases of intracranial complications dependent upon diseases of all the accessory sinuses, of which sixty-five were brain abscesses. As Freudenthal remarks, "Many cases have occurred following operations which were never reported or either not recognized antemortem."

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*Candidate's thesis which was accepted by American Laryngological, Rhinological and Otological Society, 1922.

In reporting these cases it is not with the thought of presenting something rare, but to emphasize the many and varied symptoms to which attention has often been attracted by similar reports throughout the literature.

Case 1.—On October 7th, 1920, S. P., age 16, consulted me complaining of marked headache and swelling of left upper eyelid. He gave the following history: Cold past two weeks with severe leftsided headache past week, for the past three days left upper eyelid gradually swelling until the present and eye now closed. Marked quantity of pus blown from left side of nose. Upon examination, the patient presented the following: Left eye closed with marked oedema of left upper eyelid and conjunctiva, also some puffing of the lower lid. Eye movements, pupillary reactions, and fundi were normal. Upon palpation, marked tenderness over left frontal region. Marked hypertrophy of both middle turbinates with pus flowing from beneath left middle. Transillumination showed small right frontal sinus clear, left side, large and cloudy, antra clear. X-ray pictures confirm diagnosis of left frontal sinusitis and also show left anterior ethmoids involved. Advised patient be taken to hospital immediately for nasal operation, but parents objected. On morning of October 8th, was called by family stating that boy was having convulsions and was unconscious. Upon insisting, he was taken to the hospital at once. Temperature 101, pulse 96, blood examination gave a leucocytosis of 20,000 with 79 per cent polynuclear cells. Lumbar puncture produced a clear fluid under slight increase of tension and examination showed cell count 12, globulin normal, glucose plus 3. As patient had been given a quantity of morphin to control convulsions, and was in a stupor, under a local anesthetic, removed left middle turbinate, clearing out ethmoid cells and making large opening into frontal sinus. Sphenoid opening seen clearly and no pus from same. Patient put to bed, no packing in nose. On October 9th, patient better but claims he cannot see and upon testing out vision with fingers, found he was able to see nothing but light and shadows moving before eye. Ophthalmoscopic examination of fundi, at this time, showed slight venous distention, otherwise normal. Slight tenderness in left temporal region, temperature normal. October 10th, feeling well, headache gone, and now able to see. October 11th, blood examination gave a leucocytosis of 19,000 with 90 per cent polynuclear cells, temperature 100, pulse 88. Patient drowsy when aroused, marked irritability. Morning of October 12th, nurse noticed that patient could not raise right leg, right arm, when gripping object, seemed weaker, however, able to use same. Tender place over left temporal region now fluctuating and was opened under ether. Found pus had broken through frontal sinus at external angle, also perforating the inner table, perforation being about the size of a dime. Dura appeared normal. Wound packed. October 13th, doing fine and now able to move leg. October 14th, blood examination gave a leucocytosis of 19,000 with 82 per cent polynuclear cells. October 15th, during the night had recurrence of convulsions, the first since primary operation. Reopened wound and found a small pocket of pus, as wound was healing too fast. October 16th, temperature now normal, patient doing splendidly, having complete control of both legs. Urine normal. A laboratory report from examination of the pus, as well as culture made, showed a pure culture staphylococcus aureus. This patient had been seen previously to this attack on May 28th, 1920, at which time his vision, fundi, and nose were normal. Patient last seen on November 9th, having completely regained his health, the operative scar barely visible.

Case 2.—On November 13th, 1920, Mrs. B., age 62, consulted me on account of a swelling over the right frontal region. Patient states that for the past year this swelling comes and goes and at times gives her no trouble. Examination reveals frontal sinus abscess, which has perforated outer table. X-ray picture confirms diagnosis. Advised radical operation, on which patient decided to wait. November 22nd, was called to house to see patient and found marked swelling, as well as edema of right upper eyelid. Patient stated that she had consulted another specialist, who had advised an intranasal operation, to which she had consented, but as the swelling over frontal region had increased along with the appearance of the swelling about the eye, and some headache, which until now had been absent, she became alarmed, discharging her other specialist and then sending for me. On November 23rd, at my request, consultation was asked for, and a radical operation was again advised. On November 24th, she was taken to hospital with temperature of 99, pulse 100, respiration 24. Blood examination gave a leucocytosis of 9,600 with 72 per cent polynuclears. Spinal puncture showed fluid clear and normal and no tension. On November 26th, a radical frontal sinus operation was done. Upon opening into the frontal sinus, extensive necrosis in the anterior wall was found, together with a large defect in inner table. The dura was covered with granulation tissue, although not tense or bulging. The pus found in sinus produced a pure culture of hemolyticus streptococci. The wound was not completely closed externally, but lightly packed. On November 28th, patient complained of a severe headache and blood examination gave a leucocytosis of 11,600 with 82 per cent polynuclear cells, this being the highest count shown throughout the course until pneumonia developed. Temperature 99.2, pulse 124, respiration 24. Patient continued to improve and on December 11th was sent home. On December 15th, although very little pus and wound practically healed, patient complained of severe headache. This, however, was of short duration. On account of this headache and the previous attack of headache, along with the exposed dura, I advised an exploratory operation on frontal lobe for abscess, but as patient seemed to improve and headache disappeared, family would not consent to another operation. On December 19th, patient seemed very bright, no headache, temperature normal. On December 21st, patient developed pneumonia. On December 23rd, patient began with short periods of unconsciousness, from which she could be aroused and would answer questions. December 24th, found patient had developed paralysis of left side. Again advised reopening of frontal wound and exploratory operation of frontal lobe. This, however, was refused and patient died December 27th. Autopsy not granted.

From the report of this case it will be noted that although there was a considerable pathologic process in the early stage of the disease, which had gone on for some time, outside of an occasional headache, no intracranial symptoms presented themselves, and it was not until with the development of pneumonia that the other symptoms of frontal lobe abscess became evident.

With the above cases the diagnosis was easy, due to the knowledge of the original trouble combined with the symptoms of intracranial pressure. In the true frontal lobe abscess cases, symptoms are often very misleading or hidden and the diagnosis is generally difficult.

When considering intracranial symptoms arising from cases of frontal sinusitis, one must not lose sight of the fact that we are dealing with that portion of the brain known as the silent area, the functions of which are without marked motor manifestations.

Functions are practically established for the prefrontal lobe, and the second and third frontal gyri on the left side; the former having to do with acquired skill movements, and the latter with speech and expression. The rest of this region being the great motor association regions, any irritation or pressure on any given section of it may produce sensory disturbances, due to the close linking with the postcentral region through the associational tracts. On this account, through pressure arising from the vascular system engorgement, toxic irritation or malnutrition of the association fibres and cells, do we attribute our symptoms. This is most forcibly illustrated in Case 1, in which we have the paresis of the lower opposite limb, which immediately cleared up upon evacuating the extradural abscess. The return of the convulsions, due to the pent up pus, was followed by immediate relief upon evacuating same.

From a study of the two reports, you will note that the symptom of headache is most pronounced, being too severe to be accounted for by the sinusitis alone. One may be misled unless the severity of the headache is taken into consideration, as it is far more violent in the intracranial complications, a possible explanation being the congestion of the anterior meningeal blood vessels. The normal lymph flow and oxygenation to plasma and brain cells being interfered with by the congestion of the anterior cerebral, corresponding to the superficial congestion of the meningeals, the lack of oxygen results in demand by the cells referred to the thalamus and postcentral region and is projected back as pain. Extradural type of abscesses usually causes greater headache than intradural, due to the fact that we have, as a rule, a wider involvement of the congested region. This is well seen in Case 1, where the original headache disappeared on free drainage of the frontal sinus, only to reappear in a more violent form, lasting until the epidural abscess was evacuated. In Case 2, the severe type of headache appeared late after our first operation. This, together with the appearance of the exposed dura, was the basis of our diagnosis of a cerebral abscess, at which time a second exploratory operation was advised.

In neither case was the pulse of any value, although it must not be overlooked. Many men call attention to it, laying great stress on the slow pulse, which indicates a general increased intracranial pressure.

Temperature, being one of septicemia, offers nothing characteristic. In the early stages of Case 1, relieved upon removing the source of trouble, while in Case 2, returning, when complicated with pneumonia.

In Case 1 patient complained of dizziness, which seems to be rather a rare symptom, while vomiting, although it did not occur in either case, is a great deal more frequent, and especially when occurring independent of meals, cerebral abscess must not be overlooked. This is due to the toxic or congestive irritation of the medulary center.

Referring to Case 1, it is well to note the marked symptoms of irritability and early convulsions, which at first were not localized. Disturbances of the sensorium are usually more or less marked, such as drowsiness, peevishness, weakness of memory, irritability, inclination to sleep, stupor, apathy, patient when questioned answers very slowly but correctly. These symptoms more or less marked, due to the degree of meningeal and cortical congestion.

In both cases, paresis of the opposite lower limbs was noted, Case 1 clearing up with the opening of the epidural abscess.

Optic neuritis and stasis of the papilla have been described in a number of cases, due no doubt to the increase of intracranial pressure, or possibly to the chronic passive congestion anaemia. This is well illustrated by the transient blindness which occurred in Case 1, although with the ophthalmoscope only a venous engorgement of the fundi was noticed. The external symptoms, or complications proceeding from the eyes, are usually due to the sinusitis, but oculoorbital complications seem to appear more often in those cases which are complicated by intracranial lesions and, for that reason, acquire certain diagnostic value. This being due to the anatomic relationship of the lymphvenous flow from the orbit being inward, thus conditions affecting the intracranial blood vascular system would tend to produce similar changes in the orbit.

Spinal puncture in the first case showed an increased intracranial pressure, cell count and sugar, but no bacterial growth, while in the second case it was of little value. However, in looking through the literature I find that in those cases in which the puncture is made there is usually a slight increase of pressure and the presence of a copper reducing element. Owing to the various changes which take place the value of the spinal puncture can only be estimated to any degree by the condition of the patient.

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XXXII.

ACUTE MASTOIDITIS WITH UNUSUAL COMPLICATIONS.

By E. P. Norcross, M. D.,

Chicago.

The case that I wish to report this evening presented such unusual complications that I consider it worth while to review it before the Society.

While the history of the case covers a period of several months I shall make the recital of it as brief as possible, emphasizing the unusual features.

The subject of the report is a child three and a half years of age of unusual mentality. About four weeks before I first examined her she had contracted a head cold followed by a right otitis media purulenta. A paracentesis was done followed by a profuse mucopurulent discharge. Two weeks after the paracentesis the patient had a sudden rise in the temperature and complained of pain in her head and dysphagia. Examination of the throat revealed a retropharyngeal abscess on the right side which ruptured spontaneously before the doctor had time to incise it. The child's condition then improved until the time I first saw her, one month after the onset of the otitis media.

At this time the child had a temperature of 101.6, pulse 120. She was irritable and complained of pain in the right side of her head. When she moved she held her head because her neck hurt her. There was no neck rigidity, and with the exception of a paresis of the right external rectus, no evidence of paralysis anywhere. The reflexes were normal. There was a profuse purulent discharge from the right external auditory canal and well marked tenderness over the mastoid antrum and tip. The throat was clear. The tonsils had been removed the year before.

The decision to do a simple mastoid operation at once seemed undebatable. The antrum was full of pus which extended throughout the broken down cells forming a well of pus at the tip. As the surrounding bone was hard, neither the lateral sinus nor the dura over the tegmen was exposed.

The child did not show marked improvement. The temperature became normal but the child did not regain her strength and continued to be irritable. The discharge from the external auditory canal was much less, but it did not cease. The wound did not show healthy granulations. Double vision and pain in the right eye were complained of for about a week. The W. B. C. remained at about 17,000 and the hemoglobin at 65; R. B. C., 3,500,000.

Ten days following the operation the temperature rose to 103 and the white blood count was 26,000. The child complained of pain in the neck and dysphagia. Examination of the throat disclosed another large retropharyngeal swelling, extending upwards into the postnasal space. Incisions into the mass resulted in the evacuation of about one-half ounce of thick, creamy pus. Cultures showed long chain streptococci which did not change blood. The W. B. C. dropped to 14,000.

Radiogram of the cervical spine did not show any disease of the vertebrae. The patient continued for another two weeks in fairly good condition. However, the discharge from the wound and external canal persisted. The W. B. C. again rose to 20,000.

Dr. Norval Pierce saw the patient with me and in view of the protracted course and the continued discharge the mastoid wound was reopened and a healthy dura and sinus exposed. As the cavum tympanum was filled with necrotic tissue the simple mastoid operation was converted into a radical operation.

Following this procedure the patient made rapid improvement, and was discharged from the hospital two weeks later. When last seen at my office the child was happy and good natured, submitted kindly to the ear dressing, and presented the picture of a healthy, normal child.

Three weeks later the mother telephoned to me and said that the child had become very angry about obeying some command and had had a violent spell of temper, screaming and kicking and scratching her mother. When she had been quieted, the mother had noticed that she did not seem able to call familiar objects by name. Otherwise there was no change in her condition. A careful examination was made of the reflexes and eye grounds, but nothing unusual was discovered. However, she could not or would not tell the names of familiar objects, although she knew what their use was and recognized the name when it was spoken.

She was sent back to the hospital for observation. The temperature was not elevated, but showed a tendency to be subnormal. The pulse was occasionally retarded. At times the child complained of pain in her head, more on the left side.

About ten days after entering the hospital she began to show some weakness on the right side of the body, first the facial muscles and then the leg. The grip in the hands remained about equal. The eye examination showed a tendency to convergent strabismus and diplopia, slight bilateral papillitis. There was no increase in headache or speech disturbances.

The following day there was a noticeable weakness in the right arm and while I was talking with the patient she suddenly had a convulsion involving all the extremities. She did not regain consciousness.

A trephine opening was made over the left temporal sphenoidal lobe. The brain was bulging—incision into its substance was followed by a very copious discharge of thick pus. The patient died about fourteen hours later.

A postmortem examination of the brain showed the presence of two large brain abscesses. The older and outer one, situated in the temporosphenoidal and occipital lobes, measured 10 cm. anteriorly-posteriorly and 6 cm. wide at its widest part. It reached from a point 1 cm. in front of the optic chiasm to a point 4 cm. from the tip of the occipital lobe.

A second abscess smaller and more recent in origin was situated mesially to the first described abscess. A direct connection could not be found between the abscess cavities.

This case presents several interesting features. First the occurrence of a retropharyngeal abscess which undoubtedly was caused by an infection arising in the tubal tympani cells traveling along the eustachian tube under the mucous membrane or periosteum and perichondrium to the postnasal space. In a child this distance would not be great. I believe that this is the correct explanation of the occurrence of the retropharyngeal abscess. There was no disease of the cervical spine, the throat was free from tonsils and adenoids, and the abscess ceased to form as soon as the middle ear was cleaned out.

I have not found the report of a similar case in the literature. The nearest approach is that of a case reported by P. Jacques, 1913. In this case during a mastoid operation necrotic area was found at the level of the floor of the antrum. A probe was passed about 1 cm. towards the lower angle of the sinus. Through this place enlarged by the curette an abundance of pus flowed during the following days without seemingly amending the phenomena of the general infection. There was a marked torticollis. The neck was painful upon pressure. Later dysphagia was complained of. An inspection of the throat revealed a mass in the right pharyngeal groove which, when incised, gave forth a large quantity of pus. From this day a new period began, characterized by a progressive decrease in the mastoid flow and of the phenomena of infection.

The second interesting feature is the development of a left temporal sphenoidal abscess during the course of rightsided otitis media. In reviewing the literature I have been able to find two somewhat similar cases. One reported by M. Lombard—Abscess of frontal lobe on opposite side from a chronic suppurative otitis. This patient was 28 years of age. Five years before he had been operated by Lombard for a chronic otitis media on the left side. On February 4, returning from work, the patient had an epileptic fit. The convulsions were the same on both sides; no tongue biting or emission of urine, eyes revulsed. The attack lasted five minutes. Amnesia followed. This fit was preceded for eight days by fatigue, and physical and mental depression.

February 9 he had another attack just like the first one. During the days that followed he complained of a continual headache, night and day, frontal and occipital; vertigo accompanied by nausea, both when standing and reclining; left ear painful with buzzing.

On February 14 patient in very marked stupor; no aphasia; no trouble in writing; read aloud in very loud voice; recited the multiplication table; recognized objects and named them correctly, but with hesitancy. No olfactory trouble.

Operated under chloroform. Trepanation of the temporal fossa above and a little behind the external auditory canal. Incision of the dura mater. Hypertended cerebrospinal fluid escaped. Four punctures with a sound in different directions led to nothing. Dura mater sutured with catgut.

February 15. Left hemiparesis with hypoesthesia. On left side, Babinski in extension. Abdominal cutaneous reflexes disappeared. Cremasteric diminished, patellar greatly diminished. Stupor, slow idea formation, but no aphasia; no spontaneous nystagmus, no sensation of vertigo.

February 17. Hemiparesis persisting; slight stiffness of neck; no Kernig; no temperature.

February 20. Symptoms aggravated; patient almost in coma, incontinent, answers questions wrong. Conjugate deviation of eyes toward the right. No nystagmus; temperature 37. Second operation—a few whiffs of chloroform; left vertical retroauricular incision; treparation behind sinus; incision of dura mater. Escape of cerebrospinal fluid; repeated punctures of the cerebellum; dura mater sutured; wound tamponed with gauze.

February 21. Dies in coma.

Autopsy showed abscess of right frontal lobe as large as a large prune, encapsulated; pus of the abscess was sterile.

A second case is recorded by Klesseres—an abscess in the left brain with right otitis media. The right ear drum became red and the vessels of the fundus oculi a trifle distended. The reflexes of the right knee were increased—Babinski sign present—after three days paresis of the right arm was noted. Pulse 60—temperature normal. Three days later the left cortical center was exposed; an abscess was discovered 3 cm. in circumference and the patient died of pneumonia.

The diagnosis of the left brain abscess was based upon the symptoms of paralysis of right facial muscles and right arm. The most remarkable point is the occurrence of an abscess in the left hemisphere from a right otitis media.

The formation of a brain abscess by an infected embolus from some distant focus of infection is not so uncommon. The interesting feature in the cases here reported is that the focus of infection happened to be an otitis media located on the opposite side of the head from the abscesses.

One cannot state in my case whether the child's fit of anger would favor the transmission of the embolus or whether there was present at the time of the outburst of temper a latent abscess that became active. Cases are reported of latent brain abscesses becoming active following some head injury or a mastoid operation.

Such cases as I have reported afford one material for serious thought and retrospection. It does not seem possible that an abscess of this magnitude could develop right under one's eyes, as it were, and not give definite enough symptoms upon which to base a diagnosis. There must have been a time during the course of the disease when an exploratory operation might have saved the child's life. Was the very mild aphasia in a right handed child a sufficient cause for doing such an exploration? Probably not, and yet I cannot help but feel that, had the otitis media been located in the left ear, the development of a mild aphasia would have been considered sufficient indication for reopening the mastoid wound and exploring the brain.

XXXIII.

THE ROLE OF THE TONSILS IN THE TREATMENT OF NOSE CONDITIONS, INFECTIVE AND OBSTRUCTIVE—STUDY OF 111 CASES.

By T. R. Gittins, M. D.,
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The purpose of this paper is to report a series of 111 cases of nasal sinusitis or obstruction observed and treated during the past eight months. The object has been to trace the relation of the tonsils to the end results obtained in these nose cases. These patients have been under the care of two associates and myself in private practice. All were hospital cases.

The fact that tonsils do have a great influence upon nose conditions seems so generally conceded that very little is to be found in the literature on the subject except in the discussion of sinus disease in children. Dr. L. W. Dean,[1] of Iowa City, has done a great amount of work on the latter subject in the past few years, making exhaustive studies from many angles.

Granting as we must from our clinical observations that there is some relationship, both in adults and children, between the course of nasal infection and the condition of the tonsils, these questions arise: First—Does the presence of chronic tonsils predispose the patient to nasal infections? Second—Does the development of acute tonsillitis complicate the postoperative course of the nose condition and influence the end result? Third—Does the removal of tonsils only have a beneficial effect upon the nose infection at the time or render the patient less liable to develop future rhinitis or a sinusitis? Fourth—Does the presence or absence of tonsils have a bearing on the postoperative and end results in cases where the object was the removal of nasal obstruction only?

Before considering the case reports it is well to consult the anatomy of the nose and throat with reference to the course of infection, sinuses to tonsils, or the reverse. J. K. Milne Dickie[2] in an abstract raises the question whether in acute tonsillitis following nose operations the infection passes through the lymphatics, blood stream or by direct continuity. Schönemann held that the infection passed through the lymphatics. He injected a solution of iodin into the inferior turbinates, removed the tonsils on the same side six hours later and found iodin present. Lenart and Henke tried a similar experiment with dogs and came to the same conclusion. Auerbach following similar technic found none in the tonsils. Pugnat injected an emulsion of soot into the turbinates, 24, 36 and 48 hours preceding the removal of tonsils. In 100 such cases none of the soot was demonstrated in the tonsils. He concluded that there was no direct connection between the lymphatics of the nose and throat. Along similar lines W. V. Mullin and C. T. Ryder,[3] of Colorado Springs, have reported some exhaustive work in their studies on "Lymph Drainage of the Accessory Nasal Sinuses." They experimented with injections of India ink and bacilli in the sinuses of rabbits and cats. In summing up they say, "We were struck in the first series by the absence of any discoloration of the tonsils in animals in which nearby tissues were heavily injected, and drew the inference that the tonsil perhaps has no field of absorption beyond its own extensive surface." In two cases they injected India ink into the pharyngeal mucosa adjacent to the tonsils. Autopsy here showed marked discoloration in the tissues of the pharyngeal wall but no change whatever in the tonsils.

In discussing the development of acute tonsillitis following the submucous operation Loeb[4] expresses the belief that the main factor is the length of time that the packing is retained in the nose. He advocates the removal of the nasal plug within twelve hours to decrease the prevalence of these acute throat infections.

In this series of 111 cases treated because of symptoms of nasal obstruction or sinusitis, 53 were of the former type, 58 of the latter. Included in the obstructive cases are those in which septum or turbinates, singly or together, were responsible. Twenty-six of the sinus cases were classed as acute, 32 as chronic.

The entrance examinations of the 111 cases proved that 28 had had tonsils removed previously. In the 83 cases which had tonsils present—I would prefer to call them all chronic tonsils, as who can say positively that any tonsil has not some infection—19 developed acute tonsillitis during the postoperative treatment. In 24 of the 83 cases the tonsils were removed at the time of the nose operation, thus eliminating this group from the possibility of acute tonsil infection. Here then, we have 59 postoperative nose cases, obstructive or infective with tonsils present, 19, or 32 per cent, of which developed an acute tonsillitis. As mentioned before in this paper, the theory of Loeb that acute tonsillitis develops in direct proportion to the length of time the packs are left in the nose may help to explain this high percentage. However, only in four instances did acute tonsil infection develop following the submucous resection or turbinectomy. There was no uniform method of packing adopted, the idea being to use the smallest amount of gauze or splint advisable and leaving it in place for the minimum length of time necessary to satisfactorily control the postoperative bleeding. Some of the cases had no plug, others had a small amount on one or both sides for a few hours, and still others were packed solidly on both sides for 24 hours. Certainly it is reasonable to expect that long continued packing of the nose will predispose patients to acute sinus or throat symptoms, but it is interesting and instructive to follow the even course of those cases in which the tonsils have been removed previously almost regardless of the length of time or completeness of the postoperative nasal packing.

In order better to tabulate results the submucous and turbinectomy cases are now segregated for study. There were 53 such cases in all, 34 of which had tonsils present on entrance examination. At the time of the nose operation the tonsils also were removed in 20 instances, thus leaving but 14 of the original 53 with tonsils present during the postoperative course. Four, or 28 per cent, of these 14 developed an acute tonsillitis during their convalescence. Loeb[4] quotes Horn[5] reporting a series of 110 submucous cases, 13 of which developed acute tonsillitis. One of these 13 in addition developed a severe pericarditis and endocarditis. No mention is made of the number of these cases in which the tonsils were removed previous to the nose operation. It is interesting to note here the length of time these various cases remained in the hospital. The 4 which developed acute tonsillitis averaged 8½ days; the 10 with tonsils present, 3.7 days; the 19 with tonsils absent, 3.8 days, and the 20 in which the tonsil and nose operation was done at the same time, 37 days.

A goodly percentage of this group of cases was influenced to return frequently for examination, while in other cases we received some information from follow-up letters. Of the 4 cases in Group 1 (those which developed acute tonsillitis), 3 have been followed carefully, and all have bad results—that is, they were not only not relieved of their entrance complaints but were distinctly worse than before their operation. These cases will be considered later more in detail. Of the 10 cases in Group 2 (those with tonsils present), 4 have not been heard from; 1 has a good result after six months; 1 after three months; 3 after three weeks, and 1 continues to have some difficulty in breathing and some discharge from the nose after four weeks. There were 19 in Group 3 (those with tonsils absent), with the following statistics: Four have not been heard from; 1 has a good result after six months; 3 after four months; 4 after three months; 2 after two months; 3 after one month; and 2 after two weeks. In Group 4 (those with tonsils removed at the same time as the nose operation), we find the following: Seven have not been heard from; 2 have a good result after three months; 3 after five weeks; 2 after three weeks; and 6 after two weeks. In the above statistics by good results is meant relief from the symptoms of which the patient complained on entrance examination and the development of no new symptoms. It is quite likely that a goodly percentage of those cases not heard from have a good result or we would have at least had complaints.

Returning to Group 1, we will consider these four cases in more detail.

Case 74 had a partial turbinectomy, middle each side. Two days later developed a mild tonsillitis with temperature of 99.6. No report has come from this patient after she left our care.

Case 19.—Mr. M., age 23, entered complaining of difficult breathing through nose, of long standing. No history of head colds or tonsillitis. X-rays of sinuses were negative. His diagnosis was deviated nasal septum and chronic tonsillitis. Tonsillectomy was advised but refused. On March 9th a submucous resection of the septum under local anesthesia was done. Course uneventful until March 13th, when he developed sore throat, temperature 102.6, membrane on tonsils. On the second day, or March 15th, there was much swelling of the turbinates and septal flap, thin pus was present on each side of the nose. Patient left hospital on March 17th, eight days after entrance. His general condition remained poor. He looked anemic, had poor appetite and tired easily. There continued a free discharge of pus from each side of the nose, and the swelling of the septal flap and turbinates subsided very slowly. Transillumination of antra was positive. X-ray of sinuses was refused. On April 9th, or one month following submucous operation, there was still free discharge of pus from each side of the nose, a perforation of the septum and boggy inferior turbinates. His appetite and strength were below normal and he was unable to carry on his ordinary work. When last seen, two months after his operation, he still had difficulty in breathing through his nose, much discharge back in the throat, and a chronic sore throat.

Case 30.—Mr. S., age 28, entered hospital complaining of difficult breathing through nose, no sore throat, occasional head colds. Diagnosis was deviated septum and chronic tonsillitis. Tonsillectomy was advised but refused. Submucous resection under local anesthesia was performed March 29th. March 30th, temperature was 102.4. Next day membrane over each tonsil. Fever remained above 100 for six days. Septal flap and turbinates badly swollen, almost completely obstructing nasal passages, much fullness and headaches in frontal region. Left hospital after seven days. Patient next seen in two months; still having difficulty in breathing through nose, much thick mucus from nose and back into throat. Had had some sore throat all the time since last seen. Tonsillectomy May 17th. Patient left hospital May 19th. To date has not been heard from.

Case 40.—Mr. W., age 36. Entrance complaint, difficulty in breathing through nose, frequent head colds in winter time. No sore throat or ear symptoms. Diagnosis, deviated septum and chronic tonsillitis. Tonsillectomy advised but refused. (It is our practice to advise the removal of tonsils before or at the same time in all cases where the submucous operation is performed. The probability of acute tonsillitis developing and the dangers of same are explained to the patients and relatives.) May 2nd, submucous resection under local anesthetic. March 7th, patient left hospital with septal flap healing nicely. May 9th, developed acute tonsillitis. May 10th, considerable swelling of the septal flap and inferior turbinates and some thick mucus in the nose. May 11th, developed acute otitis. Paracentesis was done. A week later ear was discharging pus freely. There was still considerable swelling of the septal mucosa and turbinates and thick mucoid discharge from each side of the nose. At this time patient passed from under our care and has not been heard from since.

Of these four cases where acute tonsillitis developed, three were followed long enough to convince us that they were not only not benefited but were distinctly worse than before they came under our care. Cases 19 and 30, with negative histories and X-rays previous to the attacks of tonsillitis, have had free discharge of pus from each side of the nose following the acute infection. Both patients refused another X-ray of the sinuses, but in Case 19 both antra were very cloudy to transillumination. Case 40 had had no previous sinus or ear symptoms, but following the attack of tonsillitis had pus in each side of the nose and an acute otitis.

Before passing from the submucous cases it is well to note that in 20 of the 53 cases the tonsils were removed at the same time the nose operation was performed. In three of these twenty the combined operations were done under local anesthetic, the remaining seventeen under ether anesthesia. Especially noticeable was the lack of shock in those patients operated under general anesthesia and the rapidity with which the nose and throat healed. Naturally, greater difficulties were encountered in doing the submucous work under general anesthesia than under local, but we feel that with care good work can be done with the former method.

Continuing the study of the cases, we find that 48 of the 111 considered in this report were operated for relief of sinus disease. Of these 48, 20 were antrum cases, 10 frontal, 17 pansinus and 1 sphenoid.

The antrum cases will be taken up first. On entrance to the hospital 16 of these 20 had tonsils present. In 4 the tonsils had been removed previously. Of the 16 with tonsils present, 7, or 43 per cent, developed an acute tonsillitis in the postoperative period; 4 were of chronic, 3 of acute sinus involvement. Eleven of the 20 antrum cases were seen in the acute stage, 9 in the chronic.

Looking at these statistics from another angle, we find that of the 11 cases which came under our care because of acute antrum disease only 1 had had the tonsils removed previously, the other 10 suffering from chronic tonsillitis at the time the acute sinus disease developed. Of the 9 cases with chronic antrum infection 3 had had the tonsils removed previous to our examination. In these latter cases, however, we have no way of determining just when the chronic sinus disease began. Might it not be possible that the chronic antrum infection was present some time before the tonsils were removed? It seems significant that only one acute antrum infection came under our care in a period of eight months, in a patient whose tonsils had been removed previously, while during the same period ten patients with a similar acute infection entered, each suffering from coincident chronic tonsillitis.

Passing on to the frontal sinus cases we find that they were 10 in number. Six of these were seen in the acute stage, 4 in the chronic. Of these 10, 8 had tonsils present on entrance examination. In contrast to the antrum cases, only 1 of the 10, or 12 per cent, developed acute tonsillitis, as against 43 per cent when we were dealing with the antrum. Of the 3 chronic frontals with tonsils present, 1 developed acute tonsillitis. Of the 5 acute frontals with tonsils, none developed acute tonsillitis.

In grouping the pansinusitis cases, we find there were 17. Seven of these were of the acute, 10 of the chronic type. On entrance, 14 of the 17 had chronic tonsils present, almost duplicating the antrum statistics. Six of this number, or 42½ per cent, developed acute tonsillitis during the postoperative course. These 6 cases were divided equally between the acute and chronic types.

Comparing with the antrum and frontal statistics, we find that of the seven patients treated for acute infection of all the sinuses on one side, all had chronic tonsils. In other words, no acute pansinus case was encountered where the patient had

had the tonsils removed previously. On the other hand, of the 10 chronic cases 3 had had the tonsils removed previous to our examination.

Only one isolated sphenoid infection came under our observation in this series of cases. This was of the acute type and had chronic tonsils present. Two days after the sphenoid was drained, this patient developed acute polyarthritis. Sinus was clear in eleven days.

Summing up, it may be significant to note that of the 24 patients who entered our services suffering from acute sinus disease, only two of the number had had the tonsils removed previously. Does this indicate possibly that patients without tonsils are more likely to escape acute sinus involvement or are we to assume that the ratio of patients with tonsils to those without is as 22 to 2?

TABULATION: SINUS CASES.

DAYS UNDER TREATMENT

		Acute			Chronic		
Case No.	*Type	Complicated by Tonsillitis	Chronic Tonsils	Tonsils Absent	Complicated by Tonsillitis	Chronic Tonsils	Tonsils Absent
1.	F				60		
2.	A					60	
4.	F			9			
5.	P	40					
6.	F						
7.	A						90
8.	F		15				
9.	A	30					
10.	A					90	
11.	A						20
12.	P					60	
15.	P		60				
16.	A					45	
18.	A			6			
20.	A						120
22.	A		15				
23.	A		30				
24.	F		21				
25.	A		24				
26.	F		15				
27.	A				21		
29.	A		14				
31.	P		15				
33.	F				30		
34.	F		10				
35.	P				45		
37.	A				45		
38.	A		8				
39.	P		90				
44.	P					120	
45.	P				80		
47.	P	130					
48.	P		14				
51.	P						14
52.	A	28					
53.	A		12				
59.	P	60					
61.	F		14				
62.	F						15
64.	P					60	
65.	A	45					
66.	P					60	
69.	P						45
87.	P						90
88.	P				180		
90.	A				30		
110.	A						
Average Days		55	23½	7½	61	70	56
No. of Cases		6	15	2	8	7	7

*F—Frontal; A—Antrum; P—Pansinus.

In a study of the above table we find a great similarity in the number of days under treatment in the chronic cases, regardless of the presence or absence of chronic tonsils or the development of an acute tonsillitis in the postoperative course. On the other hand, there is a wide variance in the statistics of the acute cases. Two patients with no tonsils present developed an acute sinus disease, and the average length of time under treatment was 7½ days. The 15 acute cases with chronic tonsils present averaged 23½ days of treatment, while in the six which developed acute tonsillitis the average time treatment was continued amounted to 55 days. These statistics suggest to us at least that where we are dealing with acute sinus disease in the presence of chronic tonsils, we may reasonably expect a longer convalescence. If acute tonsillitis comes as a complication, we may find an originally acute case becoming chronic, even when the drainage is adequate and irrigations frequent.

As was done in the submucous cases, a few of the interesting case histories will be cited in more detail.

Case 2.—Mr. C., age 35. Came to the medical service with an acute arthritis. Some pain over right face, tenderness over antrum, right antrum dark to transillumination and blurred in X-ray. Chronic tonsillitis. Teeth negative. Right antrum was drained intranasally January 5th, local anesthesia. Large quantity of thick old pus was washed out. Sinus was irrigated twice daily thereafter. On January 8th, or seven days after antrum operation, patient developed an acute tonsillitis. On January 10th, complained of pain over left antrum and a recurrence of pain in the right knee and neck muscles. On this date thick pus was irrigated from the left antrum. Five days previously this antrum was entirely negative. Patient gradually improved from this time on, but there was still considerable pain and stiffness in the joints and some pus in the antra. On January 21st, still some pus in each antrum, patient confined to bed because of arthritis. On this date tonsillectomy was done, local anesthesia. Twelve days following this operation both antra were free from pus and joints had cleared so rapidly that the patient was allowed to leave the hospital. Patient was next seen on July 5th, when he reported that all joints and muscles were feeling as good as ever. He had no discomfort. Both antra were clear at this time.

Case 5.—Mr. K., age 45. Diagnosis on January 20th, acute frontal sinusitis, right. Diseased stumps of tonsils. Right antrum negative. On this date anterior ethmoids were curetted and right frontal drained. Patient continued to have some pain over frontal sinus, some pus in nose. On January 27th, developed an acute tonsillitis. January 30th, pain over right side of face, right antrum dark to transillumination. February 1st, right antrum drained; much thick pus present. For seven days following the draining of the acute frontal in this case the antrum on that side remained clear. Three days after acute tonsillitis developed this same antrum was full of pus.

Case 37.—Mrs. R., age 29. Diagnosis, sinusitis antral acute, bilateral with otitis media supp. acute left. Chronic tonsillitis. April 10th, both antra drained intranasally. Much pus found in each side. Drum membrane incised. April 18th, still small amount of pus in each antrum. No discharge from ear. April 20th, developed acute tonsillitis. On the next day there was a recurrence of pain in the ear and it began to discharge. April 23rd, much pus in each antrum and a free discharge from the ear, temperature normal; membrane has disappeared from tonsils. April 23rd, tonsillectomy. April 27th, ear again dry. May 11th, no pus in either antrum and no return of earache or ear discharge. Patient left hospital at this time.

Case 45.—Mrs. M., age 40. Diagnosis on May 21st was ethmoiditis chronic bilateral and empyema chronic left antrum. Chronic tonsillitis. On that date ethmoids on each side and left antrum were drained intranasally. There was much pus in the antrum. May 25th, antrum clearing rapidly, only a small amount of discharge. May 26th, patient developed acute tonsillitis. Two days later antrum was again filled with thick pus. Patient was again seen on June 15th. At this time a large amount of old pus was irrigated from the antrum. When patient was last seen, on September 8th, there was still pus being irrigated from the antrum.

Case 52.—Mrs. R., aged 28. Diagnosis on June 4th, empyema acute, each antrum. Chronic tonsillitis. Antra were drained intranasally. On June 9th, developed acute tonsillitis; slow convalescence, with a gradual decrease in the amount of pus in the antra. On June 30th, antra clear.

Case 65.—Mrs. L., age 32. Diagnosis on July 11th, empyema acute right antrum. Antrum was washed out through a small trocar daily for four times. There was now no pus, so patient returned home. On July 18th, patient returned to the hospital with an acute tonsillitis. Two days later developed pain over the right antrum, and on irrigation much thick pus was found. July 21st, developed an acute suppurative otitis right. July 25th, mastoidectomy simple right and intranasal drainage of right antrum. August 25th, mastoid cavity almost healed, ear canal dry, still a small amount of pus from the right antrum. Tonsils remained subacutely inflamed. September 27th, mastoid wound healed and antrum clear. October 2nd, tontillectomy.

Case 66.—Mrs. H., age 56. Diagnosis on July 23rd, pansinusitis chronic bilateral with polyps; chronic tonsillitis. All sinuses were drained intranasally on this date. Much old pus found in each antrum. Antra were irrigated once daily, and on September 7th there was still a small amount of pus in each one. Nose was quite clean and there was a good breathing space. September 8th, developed an acute otitis media right. September 26th, still thick pus from the right ear canal, small amount of pus from right antrum. Left antrum clear. September 28th, developed acute tonsillitis. Three days later there was much pus in each antrum. At the time this record is written, October 25th, there is still much pus in each antrum.

Case 88.—Mr. S., age 26. Diagnosis on entrance, sinusitis ethmoidal and antral, chronic bilateral. Chronic tonsillitis. All sinuses left were drained intranasally. Sinuses right were not drained because patient objected. After two months of daily irrigation there was still much pus in left antrum. At this time developed acute tonsillitis. For two months both antra were irrigated practically every other day and there continued to be pus present. Tonsillectomy was done. Thirty days after removal of tonsils no pus in either antrum, nose clean. Patient has not returned since that time. It is well to note here that the sinuses on the right side were never drained except the preliminary irrigation for diagnosis, yet in thirty days after the tonsillectomy there was no pus present on either side.

Having considered the cases in which the submucous or sinus operations were done, there is left only a small group of 10 of the original 111. Three of these 10 passed from under observation before complete examinations could be made. The seven remaining patients were those with nose symptoms—that is, frequent head colds, poor nasal breathing, etc., in which only the tonsils were removed. Any nose treatment was either not advised at the time or refused by the patients. The small number of cases in this group is due to a large extent very likely to the fact that in the series of 53 submucous and turbinate cases the tonsils were removed at the time of the nose operation in 24 instances and within two weeks prior to or following on eight occasions. Thus in seven of the twenty-one remaining cases the tonsils only were removed in an attempt to relieve the nose symptoms. Of these seven, from three has come no report since their operation.

Case 94.—Mr. C., aged 34. Two years previous to examination had had middle turbinates trimmed and ethmoids drained. At the time of our examination the patient still complained of head colds and frontal headaches and occasional acute tonsillitis. March 7th, tonsillectomy was done. On September 5th, or six months later, patient reported that he had been very free from head colds or headaches. He says that his nose has given him less trouble than at any time in his life before.

Case 95.—Mr. P., age 38. Had submucous resection four years previous to our examination. Still complained of frequent head colds, poor nasal breathing and some sore throat. Examination reveals straight septum and good breathing space; chronic tonsils. Tonsillectomy, January 3rd. Patient was again seen on September 12th, or practically eight months later, and reported that he had been almost free from head colds and nose symptoms since the tonsillectomy.

Case 99.—Sister G., age 43. Entrance complaint, catches cold easily, frequent sore throats and cough. Diagnosis, deviated septum, chronic tonsillitis. June 5th, tonsillectomy. Patient was next seen September 14th, or practically eight months later, and reported much improved. She said there had been very few colds, much less nasal discharge and that she felt so much relieved that she did not wish the nose operation performed.

Case 36.—Baby O., age 2½ years. Diagnosis, otitis media suppurativa chronic left. Chronic tonsils and adenoids. There was much pus in each side of the nose. Adenoids were removed. Patient was not seen again for three weeks. Then there was in addition to the chronic otitis an acute otitis in the other ear. There was still much pus in the nose. At this time tonsils were removed. Six days after the tonsillectomy the acute ear was dry and there was much less pus in the chronic ear. Fourteen days following tonsillectomy both ears were dry and the nose was quite clean.

No further mention will be made of the sinus cases in children except to refer again to the work of Dr. L. W. Dean[1] of Iowa City. He states that 80 per cent of the chronic cases of nasal sinus disease in infants are cured by the removal of tonsils and adenoids. He attributes these results to the age of the patients, excluding long chronicity, and to comparative infrequency of nasal obstructive lesions in infancy and early childhood. So firm is his belief in the efficacy of removing tonsils and adenoids that even where pus is demonstrated in the sinuses, he practically always attends to the throat condition first, then has the patient return in a few months for further examination before any treatment of the sinus condition is advised From this method of procedure he has deducted the percentage of cures given above. To illustrate, he cites the case of an eighteen-months-old baby with a posterior cleft palate. Adenoids were removed and careful irrigation of the nose was carried out for two months. They were unable to clear the patient of a hemolytic streptococcus sinus disease. Tonsils had not been removed, because of the fear of contraction of the pillars interfering with the success of the cleft palate closure. Finally the tonsils were removed, the sinus disease disappeared rapidly and the cleft was readily closed.

SUMMARY.

1. One hundred and eleven nose cases were studied. The diagnosis in 53 instances was deviated septum or enlarged

turbinates. In 58 cases it was sinusitis. Of the sinus cases 26 were treated during the acute stage, 32 during the chronic.

2. Twenty-eight of the 111 cases had had the tonsils removed previous to entrance examination. In 24 of the remaining 83 the tonsils were removed at the time of the nose operation. Thus 59 of the original 111 patients had tonsils present during the postoperative course. Of these 59, 19, or 32 per cent, developed an acute tonsillitis following their nose operation.

3. In the 53 submucous or turbinectomy cases, 4 had tonsils present on entrance examination. In 20 instances the tonsils were removed at the time of the nose operation. Thus only 14 of the original 53 went through their postoperative course with tonsils present. Of these 14, 4, or 28 per cent, developed an acute tonsillitis.

4. In 3 of the 53 submucous or turbinectomy cases the end result was poor—that is, the patients were not only not relieved of the symptoms complained of on entrance examination but were distinctly worse than before the operation. In these three instances acute tonsillitis developed during the postoperative course.

5. Uniformly good and rapid results were obtained in cases of submucous resection where the tonsils had been removed previously or at the time of the nose operation.

6. Only two cases of acute antral and none of acute frontal sinusitis were encountered in patients whose tonsils had been removed previously. In this series acute sinus disease appeared almost always in patients suffering from acute tonsillitis.

7. In several instances acute antra developed following acute tonsillitis, complicating the course of the nose operation where the antra were clear previously. On the other hand, several acute antra resisted very active treatment until the tonsils were removed, then cleared up rapidly.

8. No frontal sinusitis case developed acute tonsillitis during the postoperative course.

9. Comparing the length of time of after-treatment in acute sinus cases, the two without tonsils averaged 7½ days, the five with tonsils averaged 23½ days, the six which developed acute tonsillitis averaged 55 days.

10. The chronic sinus cases remained under treatment practically the same number of days, regardless of the presence or absence of chronic tonsils or the development of an acute tonsillitis.

11. Quoting from Dean's statistics, probably 80 per cent of the sinus cases in infants and children are cured by the removal of tonsils and adenoids only.

12. In ten cases tonsils only were removed for the relief of nose symptoms. Four of these ten have been followed for a period of six to eight months and all have been greatly improved.

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XXXIV.

SUPPURATIONS OF THE LABYRINTH.

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HISTORY.

The development and study of labyrinthine suppuration, from the earliest experiments to the scientific studies of the present day, is a most interesting work. The studies of Flourens in 1824 to 1828 and followed later by the experiments of Ewald were the foundations upon which our present day literature is founded. Later several noted scientists, among whom are Jansen, Hinsberg and Richards, added greatly to the subject. The name of Robert Barany of Vienna perhaps stands foremost among the investigators of labyrinthine suppurations. He has conducted many experiments and has given much valuable time to the study of the labyrinth. The works of Neuman and Ruttin followed, and in this country much credit is due to further scientific study of this topic by such men as Kenison, Ballenger, Mackenzie, Friesner, Dench, Shambaugh and Duel.

ANATOMY.

That we may understand this subject better, perhaps a brief outline of the anatomy of the labyrinth would not be out of place.

The inner ear is composed essentially of two structures, (1) the osseous labyrinth, and (2) the membranous labyrinth. (1) The osseous labyrinth is made up of a series of cavities continuous with each other, namely, the cochlea, vestibule and semicircular canals. The vestibule makes up the central portion, communicating posteriorly with the semicircular canals and anteriorly with the cochlea. Posteriorly are the five apertures of the semicircular canals. Anteriorly the vestibule opens into the scala vestibuli of the cochlea.

The three semicircular canals lie posteriorly and above the vestibule, being differentiated by position, namely, the superior or anterior vertical, posterior and external or horizontal. They open by five apertures into the vestibule. The inner extremity of the anterior vertical and the upper extremity of the posterior unite and form one canal opening into the vestibule.

The cochlea is a cone shaped structure, having the base corresponding to the internal auditory meatus and apex pointing outwards and forwards. It is a sort of spirally arranged tube surrounding a central portion, the modiolus. The lamina spiralis ossea is a thin bony process which divides this spiral tube structure into two parts, the one above called the scala vestibuli and the one below the scala tympani.

(2) The membranous labyrinth, lying within the bony labyrinth, contains a fluid called endolymph, and the space between it and the real bony labyrinth is called the perilymphatic space, which contains perilymph. The utricle and saccule are the two membranous sacs in the vestibule. The former receives the endolymph from the membranous canals and the latter communicates with the membranous cochlea. All portions of the membranous labyrinth contain endolymph and communicate with each other. The ductus endolymphaticus, from the saccule uniting with the ductus utriculosaccularis from the utricle, are transmitted by the aqueductus vestibuli to communicate with the meningeal lymphatics. The inner layer of this membranous labyrinth throughout is composed of a supporting layer of cells, upon which rest the hair cells proper.

EXPERIMENTS OF EWALD.

From Ewald's experiments the following deductions are made:

(a) Excitation of a single canal produces a nystagmus only in a plane that corresponds to the plane of the canal.

(b) By reverse movements of the endolymph in any canal the induced nystagmus is reversed.

(c) The strongest nystagmus which can be induced by irritation of a single canal is always in the direction of the ear experimented upon.

A nystagmus of the vestibular origin has the following characteristics:

1. Is composed of a quick movement (quick component) in one direction and a slow movement (slow component) in the opposite direction.

2. When the eyes are turned voluntarily in the direction of the quick component, the nystagmus is increased in rapidity and in length of excursion.

3. On turning the eyes in the direction of the slow component, the nystagmus becomes very weak and at times disappears. These characteristics are usually to be elicited during the course of any acute suppurative labyrinthitis.

THE CALORIC REACTIONS.

To Barany of Vienna belongs the honor of establishing the significance and the technic of the caloric reactions and of the conclusions drawn. They are dependent on the following conditions:

1. No objective or subjective phenomena result when a normal ear is irrigated with water at ordinary body temperature.

2. Irrigating an ear with water at 70 to 78 degrees F. the following phenomena occur:

(a) A rotary nystagmus to the opposite side (towards the ear not irrigated).

(b) Patient becomes dizzy and at times nausea and vomiting may set in.

3. Irrigating an ear with water whose temperature is 110° we note the following phenomena:

(a) A rotary nystagmus to the same side (toward ear irrigated).

(b) Ataxia and vertigo and if severe enough and nausea and vomiting. I have seen patients become very much nauseated and yet never reach the vomiting stage.

The real value of all these caloric experiments is that we can determine to what extent, if any, the vestibular loss has been. This, of course, depends on the fact that if the vestibular apparatus has been impaired or destroyed, we find no caloric reactions on applying water whose temperature is 78 or 110 degrees (cold or hot).

By placing the head in the different positions one is able to thus test each semicircular canal and note its normal physiologic action.

There are two tests that stand out foremost in determining functional activity of the labyrinth. The noise apparatus given us by Barany, which enables one to determine partial or complete loss of hearing, and the caloric reaction test which reveals to us the presense or absence of static function. It is quite necessary in order to obtain true findings in the caloric reactions to have the canal free from any material, such as cholesteatomata or granulations that would inhibit the proper action of the water used. The turning tests, which are of value in certain cases, are to be used with just care, for no one knows how many cases which were in a fair degree becoming localized were by those tests converted into a diffuse suppurative condition.

The spontaneous nystagmus is really a manifest sign of acute labyrinthitis, and when the slow component is directed towards the sound side it means an overstimulated labyrinth, while if directed towards the diseased side it means either a weak or absent function. The vestibular apparatus is responsible for the slow component of the nystagmic movement.

Another important reaction, though when only positive, is the fistula test. This test should only be made when the caloric reaction appears negative. When the fistula test is positive so also one generally finds the caloric test positive. A positive fistula symptom reveals to us that the inflammatory reaction has already invaded the membranous labyrinth.

While performing a radical mastoid operation, if a fistula presents itself, it is the concensus of opinion not to probe it where there is absence of meningeal symptoms. For to do so would tend to light up a quiet process, converting a perilabyrinthitis into an active diffuse affair.

If the caloric test is positive it is better to wait until time of operation to discover the fistula. In an acute labyrinthine case, if it is determined that there is a dead labyrinth by a negative caloric and ablated hearing, it is also considered to be unwise to subject the patient to the other tests, such as whirling, turning, standing, etc. Absolute quiet is the rule in order to keep the inflammatory reaction circumscribed and to prevent the extension of same to the meninges. Nature has always been very kind to us, and here in this location she has thrown up an inflammatory wall between the meninges and the labyrinth, consequently it is to the welfare of our patient to preserve its presence.

ETIOLOGY.

As is well known, the etiology of suppurative labyrinthitis is very rarely primary. It is usually secondary to diseased conditions of the middle ear, especially otitis media suppurativa, which so frequently follows the acute exanthematous fevers, as measles, mumps and scarlet fever, particularly the latter. Suppurative labyrinthitis may follow pneumonia, meningitis, and even cases of thrombus in the petrosal sinus of the temporal bone. Undoubtedly a suppurative process might be set up as a result of meningitis, the infective agent gaining access through the lymph channels, the aqueduct of the vestibule and the cochlea. Only occasionally are suppurative processes set up in the labyrinth as a result of acute otitic infections; more commonly they occur as a sequence of long standing middle ear suppuration. They occur very frequently as an acute exacerbation of a chronic process of tympanic suppuration.

CHANNELS OF INFECTION FROM MIDDLE EAR TO LABYRINTH.

The labyrinth may become infected systemically through the blood vessels and the lymphatics, and indeed, many of those unexplainable inflammations are in all probability due to such channels. Shambaugh[1] has worked out the circulation of a guinea pig and has demonstrated that there is quite a definite anastomosis of the blood vessels of the labyrinth and those of the middle ear. It must also be remembered that toxins of a systemic disease may become localized in the labyrinth, being carried by the vessels, and thus a localized labyrinthitis becomes apparent, and soon, by lowering the vitality of the structure, makes it a ready playground for the many pyogenic bacteria which are constantly in the circulation.

There are no direct communications of the middle ear and the labyrinth. The two openings present in the osseous bony structure, foramen rotundum and foramen ovale, are covered by a dense tough membrane which stops the progress of all germs. Many different accidents may occur to cause a perforation of the outer labyrinthine wall and thus afford an easy channel of entrance to the vestibule of an existing middle ear trouble. In cases of fracture of the base of the skull through the petrous bone, microorganisms may invade the labyrinth from the nasopharynx by way of the eustachian tube and also externally through the auditory canal. During the operative attempt of extracting the stapes when necrotic, especially if in unskilled hands, the labyrinth becomes infected. Even in the removal of foreign bodies from the ear, the stapes has become dislodged and the labyrinth infected. Also during the radical mastoid operation, great care must be exercised in removing the malleus and the incus, lest the stapes should be pulled away and the membrane covering the foramen ovale become perforated.

Victor Hinsberg[2] of Breslau, after carefully searching the literature for labyrinthine suppuration complications of the middle ear, found only 198 cases which had been carefully recorded. The entrance of the infection from the middle ear to the labyrinth could be determined in only sixty-one cases of the 198. Below are given the locations as he reports them:

Fistula in horizontal semicircular canal	27 cases
Fenestra ovalis	17 cases
Fistula in posterior or superior semicircular canal	7 cases
Fistula on promontory	5 cases
Fenestra rotunda and ovalis	3 cases
Fenestra rotunda	2 cases

From the above statistics, it will be readily noticed that the infection entered the labyrinth most frequently through erosion of the horizontal semicircular canal, then through the oval window, the infection gaining entrance in only two cases through the round window.

The reason for the frequency with which the horizontal canal is involved can be ascertained on studying this from the anatomic point of view. We find

1. The horizontal or external semicircular canal protrudes somewhat upon the floor of the aditus, like a small rounded eminence.

2. Being situated in this way at a point of greatest constriction between the antrum and the tympanic cavity, it is

subject to the action of all the suppurative process of the middle ear.

3. The aditus is made up of a hard, dense, bony ring. The one exception is the horizontal canal, and this represents the weakest spot in this bony ring.

PATHOLOGY.

The pathology of suppurative labyrinthitis consists in finding the inflammatory products in the endolymph, and these may be localized or diffused throughout the vestibule and all the canals. The fibrin becomes organized, a vascularized connective tissue is formed around the entrance of the infection. If of a very virulent infection the process becomes quite extensive, and granulations may or may not be formed in the acute form. In the chronic form or where a labyrinthine infection has been set up following a long history of otitic trouble, a great many small granulations are found. Some of the canals on being opened, especially the external semicircular canal, are seen to be practically occluded by granulation growth. This vascularized connective tissue may in time be transformed into a hard, bony substance by the deposits of calcium salts. Much of the labyrinthine wall may become absorbed and broken down. Microscopically, round cell infiltration is found among the granulations, the serous membrane lining the vestibule, and the canals become congested and swollen; round cell infiltration is found in most of its walls. The footplate of the stapes may be necrosed and only a small portion left, or it may be embedded in the surrounding exudate. In one case, on opening the first and second turns of the cochlea, as is always performed in a complete labyrinthectomy, a purulent exudate was readily seen exuding from the opening. Politzer[3] states that in some cases the absorption has gone on to such an extent in the cochlea that only a small fragment of one turn was remaining and the modiolus was almost wholly gone.

The terminations of infective labyrinthitis from a pathologic viewpoint may be as follows:

(a) Complete return to the normal. The drainage being sufficient to allow most of the infective process to drain away. These cases are rare.

(b) The labyrinthine cavity may become filled by fibrous tissue. The numerous granulations present soon become permeated by connective tissue structure, and the many fibrous bands present tend to make it a hard, compact mass, which soon contracts and, in time, by deposition of calcium salts, be transformed into bone. The function of the labyrinth is much diminished in such cases.

(c) Extension of the infection to the meninges, through the aqueducts of the cochlea or vestibule; internal auditory meatus or by osteitis of the capsule from the superior semicircular canal; formation of extradural abscess, thence meningitis.

The fistulous openings in the external capsule, which are frequently found at operation or at postmortem, probably do not always represent real avenues of invasion from without inward, but at times avenues of escape through which a serous or purulent exudate is successfully discharged from the labyrinth into the tympanic cavity.

CHANNEL OF INFECTION FROM LABYRINTH TO BRAIN.

The cranial cavity frequently becomes infected by extension of the process along the acoustic nerve and the aqueducts of the cochlea and the vestibule. The infection may also extend through the internal auditory meatus, the latter being a means of common invasion. Anatomically speaking, any infection of the labyrinth may take place easier from the cranial cavity through the aqueducts of the cochlea and vestibule and through the internal auditory meatus than coming from the middle ear. For here the infection must invade and break down membranes and bones—in fact, overcome anatomic barriers to reach the labyrinth, while an infection from the dura simply has to follow well defined pathways which lead directly to the labyrinth.

SYMPTOMS.

In an acute suppurative involvement of the labyrinth the first symptoms to arise are those occurring as the result of irritation of the end filaments of the cochlear and vestibular nerves. The former by impairment or loss of hearing and tinnitus aurium, and the latter by loss of equilibrium, vertigo, nausea, vomiting and nystagmus. If the patient is not already confined to bed this forces him to go. The patient will complain of a peculiar vertigo in that all objects tend to rotate before his eyes.

A nystagmus of vestibular origin is composed of a slow movement in one direction and a quick movement in the opposite direction and rotary in character. The slow component, at the first invasion of the labyrinth, will be directed to the sound side, showing an increased functional activity of the vestibular apparatus involved. In a short time (three to four days, depending on the virulent character of the infection), the slow component will be directed to the diseased side if the vestibular apparatus has been destroyed. If the slow component is directed to the sound side it is considered evidence of some function remaining in the vestibular apparatus and would also indicate, in a slight degree, that the deafness was not entirely lost.

The nystagmus to the sound side is explained upon the fact that when the labyrinth has been destroyed on one side there is an overstimulation of Deiter's nucleus on the opposite side and hence a nystagmus to the sound side follows.

Usually the first symptoms to appear are dizziness, subjective noises and spontaneous and these in turn are the first to go, while the disturbance of equilibrium, if present, will continue for a variable time.

Kopetzky[4] holds that in labyrinthine infection the onset is found to be marked with a distinct rise in temperature and sudden appearance of facial paralysis. On the contrary, Dench holds that facial paralysis does not occur early or is a common sign of purulent labyrinthitis. Facial paralysis usually occurs in cases of long standing necrosis. As to the frequency of facial nerve involvement, many different reports are given. Ballenger[5] has found the facial nerve involved in labyrinthine suppurations in 55 per cent of the cases under his observation, while the reports of Gerber[6] and Bezold[7] vary from 77 per cent to 83 per cent. Blake[8] states that the most vulnerable points of the facial nerve are the region above the oval window and the superior portion of the posterior tympanic wall.

A gradual progressive loss of hearing developing under close observation in a comparatively short time is characteristic of inner ear involvement. In diffuse infestive labyrinthitis of long standing there is generally found a total loss of hearing.

In the acute infective labyrinthitis, after three or four days, the acute symptoms disappear, but the nystagmus remains most marked to the sound side. There is now a condition known as the latent or chronic form of infective labyrinthitis. The nystagmus continues to the sound side for about three weeks and then becomes gradually weaker and finally disappears altogether. Headache is a common accompaniment of purulent labyrinthitis. The character is that of a dull, heavy pain referred to the temporal region on the side involved.

Tinnitus aurium occurs rarely in the course of a labyrinthine suppuration and only an occasional record is found among the many cases published.

Disturbances of equilibrium are fairly constant symptoms in the acute and subacute forms. In the chronic forms they are not so constant. Such disturbauces include giddiness, standing or walking, with closed or open eyes, and vertigo when even in a position of rest. The subjective vertigo complained of by the patient is a sensation of falling.

Kerrison[9] in a study of "Vertigo of Vestibular Origin" sums up his investigations by stating "There are two distinct forms of labyrinthine vertigo and associated ataxia,—one the familiar spontaneous type, due to vestibular irritation and occurring only during the acute stage of suppurative labyrinthitis; and the other not spontaneous, not constant, not necessarily accompanied by nystagmus, characteristic only of the lateral, or quiescent, stage of the disease, and induced solely by sudden and unexpected calls upon the lost or defective oriculation-sense, in the maintenance of which the intact vestibular organs are normally so important a factor."

An intratympanic examination in certain cases may reveal evidences of necrosis of the inner wall of the tympanic cavity, if pus is found exuding from the oral window or from a fistulous track through the promontory, a positive diagnosis could be made of labyrinthine suppuration.

The daily examination of the blood should be made in every case. In the acute form a high leucocytosis and increase in polymorphonuclear percentage are found. The large mononuclears are at times increased beyond their relative count.

Frequently when a case commences to subside, an examination of the blood will detect this condition several hours prior to the time the symptoms manifest themselves. In the chronic form there is little to be learned from the blood count before the operation.

DIAGNOSIS.

The diagnosis of labyrinthine suppuration can be made fairly accurate if in the presence of a negative caloric and total loss of hearing the other typical signs present themselves —such as a spontaneous nystagmus to the opposite side, nausea and vomiting may show along with a dull headache referred generally to the ear involved.

If in an acute suppuration of the labyrinth or in an acute exacerbation of a chronic purulent otitis media, the case should develop temperature, photophobia and headache in the presence of exaggerated reflexes, positive Kernig and positive chemical changes in the spinal fluid, a positive diagnosis of beginning meningeal involvement could be made and heroic operative measures undertaken at once. In the course of an acute focal infection, if there is evidence of an acute otitis media and the above symptoms appear, one is justified in performing the simple mastoid operation at once and exercising watchful waiting before draining the labyrinth and meninges.

In the chronic labyrinthine suppurations no definite symptoms may manifest themselves. Such cases are found in examinations of chronic suppurative otitis media cases where all the regular labyrinthine tests can be safely made. Even here the fistula test should be undertaken very carefully. From the clinical history much could be gained. It is among these cases that fistulæ are found occasionally leading directly into the labyrinth. I remember several cases studied at the University Hospital, Iowa City, with Dr. L. W. Dean, which presented well marked perforations leading directly through the promontory into the vestibule.

Impaired hearing, developing while the case is under observation and later having Weber's test localized in the sound ear, is generally evidence of invasion of the labyrinth. Accompanying this, the bone conduction is much impaired, and the range of audition greatly reduced, the upper tone limit lowered and the lower tone limit elevated. Barany states that disturbance of equilibrium is characteristic only of the acute stage of labyrinthine diseases, and that it regularly disappears as the lesion advances either toward resolution or destruction of the vestibular structures. Von Stein,[16] however, holds that disturbances of equilibrium are present in all stages of suppurative labyrinthitis. When such disturbances of equilibrium are found in chronic suppurative labyrinthitis they are probably due to the overstimulation of the sound labyrinth. The caloric tests are negative. Rotary nystagmus to opposite side and the direction in which the patient would fall would always be influenced by the position of the head.

In a differential diagnosis, cerebellar abscess must be excluded. The nystagmus in a case of cerebellar abscess increases from day to day, while in suppurative labyrinthitis it becomes less and less marked and may finally disappear, owing to the extension of the suppurative process. In cerebellar abscess the quick component of the nystagmus is directed to the sound side and later it is directed to the diseased side. In labyrinthine suppuration the quick component may be directed for the first two or three days to the diseased side and then it is directed to the sound side. In cerebellar abscess the caloric tests are positive; no deafness present. Patient always tends to fall to the diseased side, irrespective of the position of the head. A vertical nystagmus is considered by some to be pathognomonic of cerebellar abscess. Choked disc may be present, whereas it seldom appears in labyrinthine suppuration. In some of those cases where the diagnosis before the mastoid has been opened is vague, they will become clear on opening into the tympanic cavity, and at this stage then and only then are some cases diagnosticated rightly.

Having excluded, by a differential diagnosis, other diseases, the diagnosis of chronic suppurative labyrinthitis can be made almost positive, when functional examination reveals total loss of hearing, negative caloric, Weber's test localized to opposite side, along with a shortened Schwabach and in addition the subjective signs of dizziness and spontaneous nystagmus. The diagnosis is confirmed when a fistula is found extending into a semicircular canal or any portion of the labyrinth.

In some few cases of localized purulent meningitis, the differential diagnosis may be difficult. A sudden rise in temperature, marked headache, vomiting, rigidity of neck muscles, positive findings in the chemical analysis of the spinal fluid, and in finding the diplococcus intracellularis meningitidis or pneumococcus in the spinal fluid points to meningeal infection.

Dench[11] states that in cases of beginning meningitis involving the vestibular nerve, where a nystagmus toward the sound side, indicating a paralysis of the affected labyrinth, may later diminish and disappear, and a nystagmus toward the affected side makes its appearance. This, he holds, is a positive evidence of a retrolabyrinthine lesion, which may be either in the cerebellar substance or in the meninges in the immediate vicinity of the vestibular nerve.

COMPLICATIONS.

The complications most liable to occur in suppurative labyrinthitis are meningitis, cerebellar abscess and septic thrombus of the jugular vein. Extension to the dura takes place through.

1. Perineural and perivascular sheaths of the seventh and eighth nerves.
2. The aqueductus vestibuli with consequent formation of an empyema of the saccus endolymphaticus and subsequent cerebellar abscess.
3. The aqueductus cochlea.
4. Erosion of superior and posterior semicircular canals.

PROGNOSIS.

It is difficult to state the prognosis of a labyrinthine suppuration. The acute form may subside gradually and healing take place. However, this may only be temporary, and sooner or later the condition returns. Every labyrinthine suppuration should be regarded as a dangerous process for the elimination of which we should use every possible means. We are not exactly able to distinguish between the favorable and the unfavorable forms. The prognosis is unfavorable in the chronic forms, perforation of the promontory wall and pressure of cholesteatoma extending into the interior of a semicircular canal.

According to Duel,[12] if the loss of function is sudden (within a few hours), following a virulent suppurative otitis media, or following a fistula test or any operative interference in the presence of perilabyrinthine inflammation, the case is probably an acute suppurative endolabyrinthitis. If such is the case there will be no return of function. On the other hand, if the function is lost slowly (days or weeks), even progressing to complete loss of both functions, there will be a final recovery or partial recovery of one or both, provided the labyrinth is not operated.

Politzer[3] holds that the prognosis is very unfavorable in an established perforation of the promontory wall, which, with a simultaneous deafness, must be looked upon as a sure sign of a diffuse labyrinthine suppuration and in which, as already mentioned, the destructive process not infrequently extends to the peripheral part of the internal meatus and to the auditory nerve.

An infection of the labyrinth of tubercular or exanthematous origin is generally held to be more destructive than due to other pyogenic causes.

TERMINATIONS.

According to Milligan and Wingrave,[13] the terminations of a labyrinthine suppuration may be as follows:

The acute diffuse pyolabyrinthitis may terminate in

1. Total destruction of the auditory function.
2. Sequestration of part or the whole of the labyrinth.
3. General blood infection.
4. Chronic disease.
5. Death.

The chronic pyolabyrinthitis may terminate in

1. Ossification of the affected area.
2. Caries of part or the whole of the labyrinth.
3. Erosion of bone and infection of the contents of the middle or posterior fossa.
4. Destruction of the static or acoustic segments of the internal ear.
5. Septicemia or pyemia.
6. Death.

TREATMENT.

There have been many different views held regarding the treatment of labyrinthine suppuration, and much confusion existed as to indications and contraindications of a labyrinthine operation. The treatment should only be considered after a most careful study of the existing conditions which are present in each individual case.

The purposes of the labyrinth operation, according to Ballenger,[14] are:

1. To prevent the extension of the purulent infection to the meninges and brain, and to check and cure an incipient meningitis having its origin in the labyrinth.

2. The second purpose is to cure an offensive purulent otorrhea which is perpetuated by a labyrinth fistula, remaining after the destruction of the labyrinth by a diffused suppurative labyrinthitis.

3. The third purpose is to relieve the patient of a distressing giddiness which occasionally persists after the cochlear nervous apparatus is destroyed by a suppurative process, while the vestibular nervous apparatus is but partially destroyed and gives rise to distressing and disabling giddiness.

The indications which are most generally accepted at the present time for opening the labyrinth are:

1. With the labyrinthine symptoms, complete deafness and nonirritability of the vestibular nerve of a chronic process or an acute exacerbation of a chronic process with beginning evidence of meningeal invasion indicate immediate opening of the labyrinth.

2. Necrosis of the labyrinth as induced by cholesteatomatous pieces discharging from the wound, and if associated with facial paralysis indicates opening of labyrinth and removal of all necrotic tissue.

3. Following a radical mastoid operation, at the first sign of meningeal irritation open and drain labyrinth.

4. According to Jansen,[15] any symptoms of cerebral complication makes a positive indication to at once open the labyrinth.

Generally speaking, there is no indication to open the labyrinth in an acute labyrinthine suppuration. However, the case must be kept under most careful observation and at the first sign of meningeal irritation the complete labyrinth operation should be performed. In the acute tpye it is better to keep the patient at absolute rest. By that I mean no movement of the head or body. Ordinarily it will not require much will power to keep such a case at rest, for the patient will remain very quiet to avoid the most distressing symptoms that arise on movement. In certain cases, with marked onset and pronounced symptoms of the process becoming diffuse the vestibule must be opened, both in front and behind the facial ridge, the cochlea uncapped and the first and second turns opened so that the meningeal fluid washes through freely. A slit is made in the dura as near the internal auditory meatus as possible. The latent stage of labyrinthine suppuration is the period in which it can be operated upon with the best prognosis. Kerrison[16] states that vomiting, headache and elevation of temperature are distinctly characteristic of vestibular irritation and are not necessarily indicative of meningeal disease, and where these are not excessive or too prolonged surgical intervention should be delayed.

In cases of chronic middle ear suppuration, where there has been evidences of past suppurative labyrinthitis, and no meningeal symptoms present, it is best and safer to open and drain the labyrinth, at the same time performing the radical mastoid operation, and the granulations, if present, should not be curetted, as this would tend to break down the barrier to a point that would open a communication with the meninges through the aqueduct of the cochlea and vestibule.

According to such men as Hinsberg, Jansen, Neumann, Barany and Richards, it is most essential that the vestibule should always be opened and drained in the chronic labyrinthine suppuration, because

1. The operation secures free drainage from the vestibule, which is always involved in suppurative labyrinthitis, and

2. Enables the operator to destroy the membranous structure of the vestibular apparatus, which is a main point in contolling permanently vestibular symptoms.

The route posterior to the facial canal from the manipulative standpoint appears to be the most convenient, because of least danger to the important structures, and allows the operator to fully expose the limits of the disease.

Much discussion has arisen during the past as to the nature and extent of the operation. Neumann[17] advocates the complete exposure of the cochlear and vestibular apparatus, while Alexander and others believe in only partial exposure. Operating during the latent stage of the disease, we are inclined to hold that a complete labyrinth operation should be performed if a fistula is found from which purulent secretion is discharging from the labyrinth or distinct evidence of necrosis is present in the vestibule or cochlea.

Ruttin has performed 100 complete labyrinth operations with one death resulting from the operation.

The primary step in every labyrinth operation is the complete exposure of the tympanic cavity and the horizontal semicircular canal by the radical mastoid operation. The internal wall of the tympanic cavity and the horizontal canal should be carefully examined for any fistulous tracks leading into the labyrinth. Should a fistulous opening be found in any of the canals they should be thoroughly opened and removal of all granulation tissue and necrotic bone. In many cases it will be necessary to chisel away the canals to their base, thus opening the vestibule above and behind. Where fistulæ are found, the labyrinth is better exposed behind the facial ridge. The posterior crus of the horizontal canal is opened and the anterior crus is chiseled away to the labyrinth. Then the bone lying below the oral window and above the round window is removed, thus exposing the vestibule and a small portion of the cochlea. The opening is now enlarged so as to freely drain the first and second turns of the cochlea. The posterior cranial fossa is not opened as a routine measure in the labyrinth operation.

Where meningeal symptoms are in evidence, then it is necessary to freely open the dura in the region of the internal auditory meatus.

If there is any evidence of remaining function in either the cochlear or vestibular apparatus, no operation upon labyrinth is justified in the absence of meningeal symptoms. The opinions differ as to the advisability of opening a dead labyrinth while performing a radical mastoid. From the recent literature and the opinions of our most experienced men, we learn that in the presence of a dead labyrinth the radical mastoid operation should only be performed unless there is direct evidence of meningeal irritation previous to the time of operating. It is pointed out elsewhere in this paper that the danger of opening a dead labyrinth in the absence of meningeal symptoms would likely convert a latent focus into an active affair which, by breaking down the barriers nature had so well established, would tend to create a beginning meningitis.

The method of Hinsberg, Bourguet and Botey are very much similar. They all reach the vestibule from in front of the facial ridge and then open and drain the promontory, etc.

Many operations have been advocated for purulent labyrinthitis, but the truth is made even more evident that we must make the surgical technic meet the desired condition in each individual case.

TWO CASES REPORTED BRIEFLY.

Case 1.—In August, 1916, Miss K., age 42, gave a history of chronic discharge from the left ear for the last seventeen years. During the last three months she has noticed peculiar dizzy spells, which forced her to bed at different times. The discharge had become more offensive, and with the advent of the dizziness had compelled her to seek relief.

Examination showed the canal full of thick purulent material, culture from which revealed a mixed infection, with the streptococcus predominating. There was a negative caloric and patient could hear loud spoken voice at two feet with Barany's apparatus in the right ear. There was a spontaneous nystagmus to the right, and Weber's test was also localized to the right ear. Temperature, 99.4; Wassermann negative, as was also general examination. No fistula or turning tests tried. Blood showed whites 9,900, polys 78 per cent.

Diagnosis: Otorrhea chronica left with acute involvement of labyrinth.

Operation.—A radical mastoid was performed, exenterating all the necrotic cells. Leading directly into the external horizontal canal was a well marked fistula, from which a seropurulent material was oozing. As there was good drainage and no symptoms of meningeal irritation it was decided not to open the vestibule or cochlea until such time as called for.

Following the operation there was a profuse drainage through the fistula from the labyrinth. This gradually diminished in quantity, becoming more fluid like. There continued an uneventful recovery. Patient was advised of the necessity of frequent observation because of the labyrinthine involvement. The spontaneous nystagmus gradually lessened and finally disappeared six weeks after the operation. Six months later the patient appeared. There was a slight moisture in the tympanic cavity, but no evidence of any further necrotic areas present. There was no dizziness, and the patient had gained some fifteen or twenty pounds in weight.

Case 2.—In March, 1918, Mr. R., age 32, farmer, came for relief of chronic suppuration in his left ear, which had troubled him for the last eleven years. At times the discharge would apparently cease and then, following a cold, it would bother him again.

Two months ago, following an attack of measles, the discharge became very thick, and he noticed for the first time that he was becoming dizzy. These attacks became more frequent and lasted longer intervals until he was barely able to be up only part of the day. On several occasions he had become very nauseated and vomiting had followed.

Examination revealed a fairly well nourished male, whose chief complaint was the chronic discharge and dizziness of the left ear. The canal was full of foul smelling pus, caloric reactions negative and absolute deafness was present. The general examination was negative. The patient was sent to the hospital to be prepared for the surgical work, and during the night his temperature suddenly rose to 103, patient unable to sleep and a marked headache was present. The fundus examination showed a congested disc and spinal puncture revealed the fluid under pressure with 2,000 cells per cubic millimeter and globulin test positive.

A diagnosis of beginning intracranial irritation was made and immediately a radical mastoid was done, and the vestibule opened, both in front and behind the facial ridge. The first and second turn of the cochlea was also uncapped. There was no evidence of any perforations in the promontory, nor were there any fistulæ found. There was a slit made in the dura close to the internal auditory meatus. For the next four days the dressings were saturated with cerebrospinal fluid. There was a spontaneous nystagmus to the right side. The patient gradually cleared up, his temperature and blood pictures gradually coming down to normal. The spontaneous nystagmus lasted four weeks. Patient, while in bed, complained of objects rotating about him. However, this phenomenon gradually cleared, and patient was discharged from the hospital some seven weeks following the operation.

Three months following this the patient reported and he was feeling much better, had gained some weight, but there was still some dizziness left.

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XXXV.

THE APPLICATION OF DENTAL MOLDING COMPOUND FOR THE MAINTENANCE OF SKIN GRAFTS IN THE MIDDLE EAR AND MASTOID CAVITIES.

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The purpose of the radical mastoid operation is known to us all, but the question arises—do we achieve the desired results, which are: the union of the external auditory canal, the middle ear, antrum and mastoid wound, into a common cavity and the lining of this cavity with epidermis through the implantation of a flap from the external canal.

The necessity for skin grafting in the middle ear and mastoid cavity as a secondary procedure, following the radical mastoid operation, is apparent.

The plastic flap, used by a large majority of otologists, works well in some cases. In the author's experience in over 100 radical mastoid operations, epidermization of the cavity, by the flap method is not always obtained. It is then that the necessity for the use of the detached graft is manifested.

The author is skeptical as to the use of the pedunculated graft of the flap operation, because pedunculated flaps must be thick. The attachment of the flap in place by sutures throws tension on the pedicle. Passive congestion may ensue, which results in death to all parts.

In dermic and epidermic detached grafts the life of the graft depends upon its contact with the underlying surface. If thorough contact is obtained and the proper surgical technic is carried out, the dangers of the graft not taking are remote. The question arises as to the securing of perfect approximation of these two surfaces. It is at this point I wish to call attention to the use of the dental molding compound.

I have used this substance in two types of cases:

First—In those cases where complete obliteration of the mastoid and middle ear cavities is desired, and,

Second—In cases in which there is a persistent otorrhea, due to infection in the eustachian tube, and when it is desired to obliterate the middle ear orifice of this tube.

In the first instance, when it is desired to completely obliterate the mastoid and middle ear cavities, after having the wound prepared for the reception of a skin graft, a sheet of sterile dental molding compound is immersed in hot water for a period of thirty seconds, when it is taken out and allowed to cool, so that it remains flexible though not gummy. The compound is then packed into the mastoid cavity and middle ear and allowed to remain until it hardens, when it is removed.

With this impression of the cavity, the graft is then taken, preferably from the thigh, taking enough epidermis to cover the cavities. It is then spread over the impression compound, raw surface outward, seeing that there is no turning in of the edges. This done, the molding compound, with the graft lying over it, is inserted into the mastoid wound and firm pressure is made, so that it will slip into place and approximation of the surfaces obtained.

The mastoid wound is allowed to remain open. A gauze pad is placed over it. Drainage is obtained through the external auditory canal by a gauze wick, and a pressure bandage is applied to the mastoid wound. The bandage is removed in four days, and the pressure compound taken out, and the wound is closed under local anesthesia.

The second variety of cases, as before stated, are those where the disease is confined to the eustachian tube, and after thorough curettage it is desired to obliterate the orifice of the tube by skin graft.

For this type of case, the writer introduces the impression compound through the external canal into the middle ear cavity, taking an impression of the part of the cavity that it is desired to obliterate, namely, the eustachian orifice. A skin graft is taken and placed over the impression compound, raw surface outward. It is then gently introduced through the external canal into the middle ear cavity. Firm pressure is made, so that approximation of surfaces is obtained. The compound is held in place by cotton packs, together with a pressure bandage over the external canal.

After four days the bandage is cut, and the impression compound gently drawn out of the external canal with a pair of forceps.

CONCLUSION.

The success of a skin graft, in no matter what part of the body, depends upon approximation of the surfaces. This can only be brought about by pressure. It is for this reason that the author has used this method of procedure.

403-407 Carter Building.

XXXVI.

SOME REMARKS ON THE INDICATIONS FOR SIMPLE MASTOID OPERATION IN THE ADULT.

By John B. Rae, M. B., C. M.,

New York.

We arrive at a diagnosis of mastoiditis on information which we receive from three sources: (1) The general or constitutional symptoms; (2) the information which we receive from the ear itself; (3) the laboratory aids.

The temperature in uncomplicated mastoiditis is low and may be normal, even in the worst cases. There is general malaise, loss of appetite and coated tongue. Pain may or may not be an evident symptom. When present it is referred to the temporal region, is dull and aching in character, is worse at night and sometimes disturbs or prevents sleep. Pain may also be entirely absent.

On inspection of the canal and drum head, we will find an acute or chronic otitis media, or there will be evidence or history of a recent involvement of the middle ear. It is generally conceded that a purulent middle ear always precedes a pus mastoid, even although the history may be indefinite and the local appearances little suggestive. The location and size of the perforation in the drum head, should that be present, will be noted, and its adequacy or inadequacy for drainage will be considered. An effort should be made to estimate the amount of discharge. This is done by wiping out the canal. The patient is then directed to Valsalva, and the amount of pus forced through the opening into the canal should be noted. This procedure may be repeated until the middle ear cavity is emptied, and in this way an estimation of the amount of discharge may be arrived at, at one sitting. This estimation of the amount of discharge is important, as in the absence of other symptoms this may be the deciding factor in determining operation. Acute suppression of discharge with increase of pain and temperature is also quite significant. Presence or absence of prolapse of the posterosuperior canal wall should be looked for. This is an edema at the junction of canal wall and drum head. The acute fold at the junction is lost, and it is impossible to say where the canal wall terminates and drum head begins. It is most likely due to swelling of the periosteum caused by involvement of cells in the neighborhood of the posterior canal wall or of the canal wall itself.

Swelling behind the ear is a late symptom of mastoiditis in the adult. This swelling may be simple edema or subperiosteal abscess. Needless to say, in either case this would be an indication for operation in the adult. Swelling behind the ear is not always due to mastoiditis and will call for a differential diagnosis between that due to mastoiditis, to furunculosis and to local cellulitis.

When slight in amount postauricular edema is detected by standing behind the patient and observing both ears at the same time. Obliteration of the postauricular fold can be thus observed, even though displacement of the auricle is scarcely perceptible otherwise.

Perhaps the most constant symptom of mastoiditis is tenderness on pressure. This is elicited by firm pressure inwards and backwards over various points of the mastoid process. Care must be taken that no movement of the cartilage is made, as this will cause great pain if we have a furuncle in the canal. Tenderness on pressure is due to direct compression of the inflamed bone and not to periostitis, which is a late modification. Tenderness is as a rule an early symptom. The extent of the tenderness is mainly determined by the character of the mastoid. It is elicited over the antrum, over the tip and over the whole surface of the mastoid if this is very cellular and involved. It is a mistake to speak of tenderness over the mastoid emissary vein, because we can never be certain of the location of this vessel. Tenderness on pressure may be entirely absent. This may be due to an excessively thick cortex, to a very forward sinus, leaving little space for cells, or to sclerosis of the entire mastoid process.

The laboratory aids have references to the identification of the infecting organism, to the total and differential blood count, to the blood culture and to the X-ray findings.

While it may be of some value to know the causative organism, too much stress should not be laid on this. Clinical observation of the progress of the disease is much more important. If we presume, for example, that a staphylococcus infection is necessarily a mild one, we may adopt an attitude with regard to a particular case that is not at all justified by its clinical course. It is the experience of most of us that any infecting organism may in any individual case give rise to the greatest destruction. It is therefore wise to emphasize the importance of clinical observation while by no means minimizing the value of the laboratory aids.

The blood counts and culture are not of any great value in uncomplicated mastoid cases. It is only when the deeper structures are involved or threatened that they are really called for.

It is equally true that there is usually no need for X-ray examination in the average case of acute mastoiditis. Apart from the information which the picture gives to the observer, the exhibition of the X-ray plate to the patient or his friend may have considerable influence in procuring permission for operation.

In the X-ray examinations there should be the closest cooperation between the otologist and the radiologist. The latter should receive from the otologist sufficient information about the patient to give him an intelligent idea of the clinical situation. The picture having been taken, both observers should make a record of their interpretations and these should always be checked up in cases which come to operation. In this way a just estimate of the value of this aid to diagnosis will be reached and both clinician and technician enabled to interpret the pictures to the advantage of the patient. We have not yet arrived at the point at which pictures can always be absolutely interpreted, and too often do we find that the report is ambiguous and undecided in those very cases in which we had hoped that the X-ray might prove to be a decided factor and enable us to set a definite course.

With all or a majority of these many symptoms present, the diagnosis of mastoiditis is not difficult, nor can the indication for operation be missed when they progress or even continue without increase, over a period of a few days. If asked to state in a word the indication for operation, the writer would answer that operation is indicated as soon as we are assured of the presence of pus in the mastoid. He can see no reason for delay once that conclusion has been reached.

It must be remembered that in a number of cases in which we have to deal with a mastoiditis complicating an acute middle ear, a distinct improvement occurs in all the symptoms when a myringotomy has been performed and treatment started with the patient at rest in bed. Pain subsides, temperature approaches or reaches normal, and the mastoid operation may have to be recommended in spite of this apparent improvement.

We must also be on our guard against the natural tendency of a patient to minimize or hide tenderness on pressure. This applies particularly to observation cases in hospital who have had the chance to see other patients returned from the operating room and to witness dressings: They are anxious to avoid operation and will sometimes do their utmost to delude themselves and deceive the examiner.

In addition there can be no doubt that in unoperated cases of mastoiditis the patient may rapidly acquire a complete tolerance. It is not an uncommon experience to find a patient who has been attending to his every day affairs over a period of weeks without especially complaining of his mastoid, which has been slowly going from bad to worse.

It is in the cases of quiet mastoiditis in which most of the classical sympotms are lacking, that the judgment, firmness and courage of the otologist will be called for. With the temperature normal and the patient free from pain, the operative indications may be very limited in number, and apparent and urgent only to the otologist and not at all so to the patient and his friends. Needless to say, the quiet case is particularly urgent, because of the danger of the onset of complications during a period of procrastination, which to the unskilled observer seems absolutely justifiable. The X-ray here offers its best field of usefulness, and if the findings are positive immediate operation should be urged.

The clinical evidence in these quiet cases has to do with the appearance in the canal, the amount and character of the discharge, the presence of persistent tenderness on pressure or, less frequently, the persistence of an otherwise unaccountable temperature. It is also quite noteworthy that the tongue often remains coated. With regard to the canal we find marked prolapse of the posterosuperior canal wall or more frequently a progressive narrowing of the entire fundus. The visible drum area is quite small, and if discharge be present it may show as a small pulsating bead at the inadequate opening in the drum membrane. Parenthetically it may be stated that these are the cases in which repeated myringotomies are sometimes performed. It is a safe general statement that when one or two myringotomies, made under favorable conditions, fail to secure adequate drainage and bring about relief, that opening of the antrum is a really conservative measure and that this is indicated rather than further myringotomies.

In those cases in which excessive amount of discharge is the characteristic symptom, the pus may be thick and creamy and adhering to all the canal walls so that it fills the lumen of the speculum when introduced. When wiped out the canal will fill up again immediately, when the patient is directed to valsalva. We conclude that this amount of pus is not coming from the middle ear alone.

Tenderness on pressure when present does not differ in location or degree from that described a few minutes ago, and depends on the character of the mastoid itself and the extent of involvement.

We must not forget that in the quiet type being described the decision for or against operation may have to be made not in the presence of all of these groups of symptoms but in the presence of only one. And also that the general condition and feeling of the patient are such that the gravity of the situation is by no means apparent to the patient himself, to his friends or even to his general medical adviser.

In conclusion, then, in the average case in which all the classical symptoms continue or increase for a few days after myringotomy and treatment, we may be reasonably certain of the presence of pus in the mastoid and advise operation.

It is the opinion of the writer that no definite limit in days should be set down as a safe observation period, but that each case should be considered on its merits, particularly from the strictly clinical standpoint, and with regard to increasing or decreasing severity of the symptoms and limitation or extension of the pathologic process in the bone. The average case allows plenty of time, say up to a week, for observation in the absence of any urgent call, and experience proves that cases so held over do better after operation when nature has had a chance to collect the constitutional and local resources in the fight against the disease.

In the quiet cases an X-ray is immediately called for. Should this show bone destruction or be otherwise positive, immediate operation is indicated. The contrast between the healthy and the diseased side may prove to the patient that his condition is more serious than he had imagined. It is a constant source of astonishment to find the amount of destruction that has taken place in these cases with a minimum of symptoms.

Should the X-ray be indefinite, operation should still be urged.

It is often observed in these doubtful cases that the hearing is diminished out of proportion to the apparent lesion, and this is of considerable diagnostic value.

It seems to the writer that the correct attitude of the otologist in face of a quiet mastoid should be to ignore the negatives, to dismiss from his mind what is lacking in the symptomatology and to concentrate on what is positive, even should that be but one lone symptom.

The writer has yet to regret opening a single quiet mastoid in which the average observer has made the clinical diagnosis.

XXXVII.

SPECIMENS ILLUSTRATING OPERATIVE AURAL SURGERY AND THE MAKING BY GRADUATE STUDENTS OF PREPARATIONS SHOWING AURAL SURGICAL ANATOMY.

By E. B. Gleason, M. D.,

Philadelphia.

The writing of this paper was inspired by the statement of George E. Shambaugh in his introduction to the chapter on the surgical anatomy of the ear in Loeb's "Operative Surgery of the Nose, Throat and Ear." "The first problem for the surgeon who would undertake the operations on the ear is to master the details of the anatomy of this region. This cannot be acquired from textbooks, nor is this knowledge readily gained by attempts to do these operations on the cadaver. A thorough grasp of the complicated anatomy of the temporal bone is best acquired by the study of preparations made especially to show this or that relation. The knowledge comes through the actual making and handling of such preparations."

As to preparations to be actually handled by students in illustrating a cadaver course on the surgery of the ear, a minimum outfit should consist of specimens showing each of the more common operations, either on macerated and bleached skulls, dried heads or wet preparations, supplemented by anatomic preparations on detached temporal bones showing the more common anomalies. Briefly it is as follows:

1. The temporal bone of an infant at birth, separated into the annulus, ossicles, squamous and petrous portions.

2. The simple and radical operation on the skull of an infant at birth.

Supplementary specimens. 2A. Malleus, incus, stapes; 2B. Malleoincudal articulation on the "scute." 2C. Ankylosis of this articulation.

These specimens show the ossicles, tympanum, antrum and internal ear of practically the same size as in the adult, developmental changes being due to the growth of the mastoid process with the formation of pneumatic cells, should these form; and to the growth of the external auditory canal (tympanic bone), resulting in a changed position of the stylomastoid foramen. It is no longer on the external surface of the skull as in the infant. Consequently, the lower portion of the primary incision of the mastoid operation should be further back than in an adult. The antrum is somewhat posterior to the posterior tubercle of the annulus and between it and the linea temporalis.

Preparations illustrating ear operations are better shown upon a half head or whole skull, from which the calvaria has been removed, than on detached temporal bones, because the average student has difficulty in visualizing the relationship of a detached temporal bone to the skull. He usually holds a temporal bone as if the squama and inner wall of the tympanum were vertical and the facial canal sloped downward, backward and outward from it, while in the skull the descending portion of the facial canal is always vertical and the plane of the inner wall of the tympanum is downward, inward and forward. Hence the antrum is always more superficial than the rest of the tympanum.

The specimens should be of such a character as not to be readily injured by rough handling or contact with dirt. They are supposed to be handed around while students are at work and occasionally dropped on the floor and otherwise maltreated.

LANDMARKS FOR THE MASTOID OPERATIONS.

3. In this skull, a portion of the descending part of the facial canal is laid open and tinted red; the contour of the surrounding parts has been preserved as much as possible, particular pains being taken to preserve the pyramid that contained the stapedius muscle in order to show what portion of the inner wall of the canal can be removed in the radical mastoid operation without endangering the facial nerve. The posterior portion of the prominence of the horizontal semicircular canal has been uncapped in the position where it is most frequently laid open by a careless operator in mastoid operations. On the other side of the specimen the superficial landmarks for the

mastoid operations are shown, and the semicircular canals laid open and the tegmen removed. The line of the descending portion of the facial nerve is a straight vertical line between the stylomastoid foramen and a point just posterior to the oval window. The root of the styloid process can usually be felt with the finger tip during a mastoid operation. Under such circumstances, the stylomastoid foramen lies immediately beneath the finger tip, and if in a radical mastoid operation, when the stapes can be seen, the line of the facial nerve is a straight line between the finger tip and a point immediately posterior to the stapes. A very important part of a cadaver course is the exposure of the horizontal and vertical part of the facial curve. The cleavage planes of the bone are such that the vertical portion can be laid bare by two strokes of the chisel so directed as to remove a triangular piece of bone between the oval window and the stylomastoid foramen; but by a student the bone is much better removed layer by layer; the curve of the nerve downward behind the oval window where the bone over the nerve is very thin being the starting point and principal landmark for the dissection.

Supplementary Specimens.— 3A. Superficial landmarks. 3B. Section parallel to the meatus in a small celled pneumatic mastoid. The tegmen has been removed to show the position of the "boss" of the external semicircular canal, the ossicles and other "internal" landmarks.

3C. Radical mastoid with horizontal and vertical portions of the facial nerve laid open. Both the external and the posterior semicircular canals are laid open. Part of the tegmen has been removed and the dura exposed. Nearly all the accidents that could happen to an unfortunate operator in a mastoid operation are shown in the specimen.

3D. This specimen shows an extremely superficial and anterior position of the sigmoid sinus, so that it would be difficult to expose the antrum by the usual method without wounding the sinus. The bone is of the infantile type and the antrum very small. (See also 4A and 4B.)

THE MASTOID OPERATIONS.

4. A skull on one side of which a simple mastoid operation has been done and on the other a radical. A comparison of

this specimen with the dissection of the facial nerve in No. 3 shows how much of the wall between the tympanum and the artificial cavity in the bone it is safe to remove in a radical mastoid operation. At the inner portion it is rather greater than would appear safe to the uninitiated.

Supplementary Specimens.—4A. The Stacke's operation. From this operation, by combination with the simple mastoid operation, was developed the modern radical mastoid operation. A modification of Stacke's operation is occasionally performed at the present time in connection with the removal of the ossicles in chronic otorrhea.

4B and 4C. These specimens illustrate the difficulty of exposing the antrum when the sigmoid sinus is superficial and far forward. In both the specimens the antrum is of normal size. However, the difficulty of reaching the antrum is not as great as in specimen 3D.

THE SIGMOID SINUS, OTIC BRAIN ABSCESS AND INTERNAL EAR.

5. One side of this skull shows the operation for exposing and opening the sigmoid portion of the lateral sinus. The operation has been done on a dried head, the dura remaining intact. The other side of the head shows a Jansen-Neumann operation and a detached temporal bone S. A., the simpler Hinsburg operation. Both specimens show the removal of the tegmen and the removal of the bone of Troutman's triangle, with exposure of the dura as in the search for a suspected brain abscess in these localities.

Supplementary Specimens.—5A. 1. Temporosphenoidal abscess. 2. Jansen-Neumann operation without exposing the dura. 3. Removal of bone for temporosphenoidal abscess. 4. Perisinus abscess. 5B. Hinsburg operation.

THE INTERNAL EAR.

6. A skull with the semicircular canals of both ears laid open.

Supplementary specimens:

6A. Specimen showing the proximity of the internal carotid to the anterior portion of the first turn of the cochlea and the eustachian tube.

6B. Topography of the horizontal portion of the facial nerve in relation to the vestibule and cochlea.

6C. Vertical portion of the facial nerve.

6D. The vestibule, showing the position of the ampulla and indifferent ends of the semicircular canals.

PREPARATION OF SPECIMENS SHOWING THE SURGICAL ANATOMY OF THE EAR BY GRADUATE STUDENTS.

As in dentistry a candidate for the degree of D. D. S. is required to present a specimen or his skill in making artificial dentures, so a graduate student should be required to present specimens of his work showing the surgical anatomy of the ear. In the dead room a work bench, motor, burrs, and other tools and instruments necessary for the purpose should be at his disposal.

He should be encouraged to produce specimens rivaling in beautiful display of anatomic details those imported from France, but at present nothing more is contemplated than the production of specimens showing the structures of most surgical importance in the more common operations upon the ear.

7. This specimen is especially recommended as an example of this type. The anterior wall of the external auditory canal has been removed to show the external surface of the drum head. The posterior portion of the internal meatus has been removed to afford a view of its fundus. The tegmen has been removed and shows the antrum with the cellular structure of the bone surrounding the antrum The inner surface of the drum head, ossicles and muscles are plainly in view. Trautman's triangle and the capsule of the labyrinth are shown. The capsule is laid open in such a manner as to show the vestibule, semicircular canals, cochlea, the first part of the course of the facial nerve within the temporal bone and the geniculate ganglion. The surgical relations of the sigmoid, inferior and superior petrosal sinuses and the bulb of the jugular vein are shown. A sufficient amount of the upper portion of the carotid canal is removed to show the proximity of the internal carotid to the cochlea and eustachian tube.

8. Is a similar dissection of the macerated and bleached temporal bone.

9. Is a dissection made in such a manner as to show better than specimens 7 and 8 the course of the facial nerve and the relation of the cochlea, vestibule, horizontal and superior semicircular canals to the inner wall of the tympanum. All of these structures are laid open but the fenestra remain intact. It is possible for a student to make either of the specimens 7, 8, 9 in two or three hours; but a longer time is well spent, as a familiarity with the important anatomic structures of the temporal bone and their relationship to each other is best acquired by the slow and careful making of such specimens. It is probable that specimens made with a burr are superior to sections made with a saw for learning the anatomy of the parts. However the methods can sometimes be combined to advantage, as in 9 and 10. The most useful sections for displaying important anatomic structures are vertical and horizontal sections through the external and internal auditory meati; and vertical sections through the tubotympanic axis and through the petrous bone parallel to the tubotympanic axis.

10. Is a section through the tubotympanic axis enabling the worker to readily open the labyrinth from the inner wall of the tympanum. Trautman's triangle has been partly removed in order to better show the capsule of the labyrinth. This is laid open. The fenestra are left intact. The horizontal and vertical portions of the facial canal are better shown in this specimen than in most of the others.

Dr. Frank A. Bridgett has kindly loaned for inspection some of the specimens he made during his cadaver course a year ago.

SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of December 5, 1921.

THE PRESIDENT, DR. ROBERT SONNENSCHEIN, PRESIDING.

Epithelioma of the Tongue.

DR. ALFRED LEWY showed a patient who had first been seen by him in the latter part of September, 1920. He had complained for two months of discomfort, rather than actual pain, at the right side of the root of the tongue, had lost some weight and did not feel quite as well as usual. Inspection showed three raspberry-like prominences, with an irregular fissure, situated at the right side and dorsum of the tongue, adjacent to the right tonsil, and some infiltration of the right posterior pillar. Examination for tuberculosis and syphilis being negative, he proceeded on October 9th to coagulate the entire area, using the D'Arsonal current, about 1200 m. a. The slough was separated in about two weeks, the patient suffering more or less pain in the meantime. Histologic section of tissue, made at the time of operation, was exhibited and showed the epithelium invading the underlying tissue at one point.

Several weeks after the operation the gland bearing tissue of the right side of the neck was removed. Examination of this glandular tissue revealed no regional extension of the epithelioma. The patient was subsequently X-rayed. Following this the operated region in the side of the neck became hard and swollen, but this had since subsided to a slight infiltration.

Several rough teeth had been extracted, and the patient wore a little hard rubber prothesis to protect the edge of the tongue from friction. There appeared to be a mild grade of parasthesia of the tip of the tongue.

He had gained in weight and felt very well. His general appearance was excellent. He had been taking intermittently a colloidal preparation of arsenic.

Dr. Lewy stated that in another case there was a carcinoma of the tonsil, which he operated by the same method as that used in the case he showed. That patient remained well for nine months and died of metastases. There was no recurrence in situ, and in that case the pain was relieved immediately. There was a large slough in the throat, which separated without trouble. The other two cases went on from bad to worse without any benefit whatever.

Bilateral Labyrinthitis Following Gonorrhea.

Dr. George W. Boot presented a patient with bilateral labyrinthitis following gonorrhea. This patient, a white man of about forty, had had a chronic gonorrheal urethritis and prostatitis for about a year. On August 10, 1921, while under treatment for the urethritis he complained of deafness. On October 8th, he complained of head noises. On October 13th he was unable to hear any of the tuning forks by either air or bone conduction. On that date there was no spontaneous past pointing. He had a marked swelling in the lumbar region, which was red and angry looking, and the whole region was much infiltrated. X-ray examination of the back was not satisfactory. On November 6th, deep fluctuation of the mass in the lumbar region was detected, but attempts at aspiration were unsuccessful. Rotation tests on November 8th gave the following: No spontaneous past pointing; after rotation to the right, momentary nystagmus only; no past pointing; after rotation to the left, momentary nystagmus only; momentary past pointing.

Diagnosis: Chronic gonorrheal urethritis; osteoarthritis of the lumbar spine; total labyrinthine deafness on both sides with total loss of vestibular function.

The Wassermann reaction was negative. A small swelling on the forehead which suggested gumma proved on excision to be a small lipoma. In spite of the total loss of function of both vestibular systems, the patient had never been markedly dizzy and had never been unable to walk. The symptoms connected with the eighth nerve ran their course in about a month.

Whether the rapid labyrinthitis was due to a gonorrheal infection of the labyrinths, Dr. Boot was unable to say, but thought it seemed quite as likely as anything else, and in view of the known occurrence of gonorrheal arthritis, gonorrheal iritis and gonorrheal spurs on the heel, it seemed to him more likely than that the labyrinthitis should be due to an accompanying infection by other pus organisms.

Dr. Boot also presented a patient with a tumor of the left vagus. This was a colored man, forty years of age. His illness began about February 15, 1917, as a sensation of pressure about the neck and upper part of the trunk. This was followed by a dry strangling cough, difficult breathing and often blindness and unconsciousness. The unconscious spells were accompanied or preceded by convulsions and had occurred as often as ten times an hour. The spells were particularly apt to occur with the approach of sleep, and the patient felt as if he were choking to death. On admission to Cook County Hospital his complaint was of pain on left side of neck and cramps of the muscles of the left side of neck.

He had malaria in 1908, gonorrhea in 1911 and an aural discharge in 1901. He had been married sixteen years and has two children alive and well. There had been no deaths in his family. He had vomited occasionally and had lost twenty-five pounds in the last six months. He has a cough before the onset of a convulsion. Examination of the neck showed a small tumor mass about 1 cm. in diameter lying just anterior to the sternomastoid muscle, opposite the upper part of the larynx. This tumor was firm, movable and apparently lay inside the carotid sheath. Pressure on it caused pain and cough. If firm pressure was continued the pulse slowed markedly and on auscultation the heart sounds disappeared. A convulsion then appeared. Examination of the larynx showed the left vocal cord to be paralyzed in adduction. This patient had a fibroid tumor of the left vagus with abductor paralysis of the larynx and the Stokes-Adams syndrome. The Stokes-Adams syndrome could be produced by pressure at any time. (The accompanying pulse tracing shows how pressure on the tumor caused slowing of the pulse.)

DISCUSSION.

Dr. J. Holinger was interested in the first case because of the finding of labyrinthitis of gonorrheal nature. Labyrinthitis from gonorrhea was never heard of. In his opinion, a labyrinthitis would have much more stormy symptoms than those present in this case. He believed the complement fixation test would show whether there were toxins or an acoustic neuritis. He thought the tumor on the forehead was a gumma. It looked and felt like a gumma and was in the typical position. The fact that the Wassermann reaction was negative proved nothing and he advised the use of injections of arsphenamin. He believed the case had nothing to do with gonorrhea but was a typical syphilitic deafness.

DR. ALFRED LEWY thought Dr. Holinger's explanation was the feasible one. They had not excluded syphilis. It might also be possible for bony changes at the internal auditory canals to shut off both nerves.

"The Efficiency of Artificial Aids to Hearing."

DR. PAUL E. SABINE, Riverbank Laboratories, Geneva, Illinois (by invitation), presented a paper on "The Efficiency of Artificial Aids to Hearing."

ABSTRACT.

This paper gave the results of tests made upon eleven different devices for the aid of the deaf, made at the Wallace Clement Sabine Laboratory, Riverbank, Geneva, Illinois. The method employed was a modification of that developed by Professor Wallace C. Sabine for the study of problems in architectural acoustics.* The observations were made by a deaf observer, the cause of deafness, as diagnosed by Dr. J. Gordon Wilson, being fixation of the stapes with no involvement of the inner ear. Sound from a set of calibrated organ pipes was produced in a large empty room. The time required for the sound, as it died away after the pipe had ceased to speak, to fall to the threshold of audibility was determined. Under the same conditions this time was measured when the particular device being tested was applied to the ear of the observer. From the difference of these times, the amplification produced by the instrument was computed. The experiments covered the range of tones from 128 to 4096 double vibrations.

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*See Proc. of the American Academy of Science, Vol. XLII, No. 2, June, 1906.

Lantern slides were shown giving the amplification for the various tones tried. For instruments of the open trumpet type these amplifications were found to follow the order of size of the instruments. The effect of the natural tone of the air column enclosed by the walls of the trumpet were shown in increased amplification of these tones. The maximum amplification produced by the largest instrument tried was about twenty fold. Instruments of the trumpet type, modified with the view of reflecting and focusing the sound, gave no greater magnifications than open trumpets of the same size. The method employed in these tests did not permit of conclusions being drawn as to the efficiency of speaking tubes.

Instruments in which the vibrations of diaphragms held in contact with the teeth and thence, by bone conduction, are carried to the auditory nerve, were found to produce positive, though slight amplifications for low pitched sounds, but were ineffective in the case of tones above middle C.

Telephone devices produced relatively large amplifications and showed a marked increase in efficiency for tones near the natural frequency of vibration of the transmitter and receiver diaphragms. Thus one device gave a magnification of three hundred fold for tones in the neighborhood of 100 vibrations per second, which was the natural frequency of the transmitter diaphragm.

By comparison with the duration of sound audible to normal ears under identical conditions, the amplification necessary to give the ear of the deaf observer a sensitivity equal to that of normal hearing was computed. This varied with the different tones but was of the order of 100,000.

It was pointed out that the discrepancy between the amplification produced by the best of the hearing devices and that required to produce normal sensitivity does not argue the entire inadequacy of hearing aids. Measurements show that sounds of ordinary loudness, such as speech sounds, for example, are of the order of 1,000,000 times the threshold intensity. Hence a hearing device which produces an amplification of 300 fold without distorting the quality of the sound and without the extraneous sounds occurring in telephonic devices would prove an extremely valuable aid in any but extreme cases of deafness, even though the deaf ear thus aided would be much less sensitive to faint sounds than are normal ears.

Dr. J. Gordon Wilson and Dr. John P. Minton presented a paper entitled:

"The Minimum Audibility of Hearing in Normal and Defective Ears."

ABSTRACT.

"Only within the last few years has there been any marked attempt to study deficiency of hearing by the use of precise physical measurements carried on by a physicist in cooperation with an otologist. The work which the writers have been carrying on during the past two years is an example of such a cooperation, which it is hoped will be productive of an increase in our knowledge, physical and physiologic, of audition. The correlation of data, taken by a physicist and by an otologist, promises to yield results of fundamental importance and of practical value. This work has been made possible through the development during recent years of the vacuum tubes and through the increased knowledge of telephone receivers.

"Experimental Method.—The audion oscillator, a telephone receiver tuned to a natural period above the range over which the tests are being made, and a Wheatstone bridge circuit are employed to make measurements of the current which makes audible tonal vibrations in the telephone receiver. The electrical and the vibrational characteristics of the receivers are determined so that the vibrational energy of the diaphragm can be calculated for ears with normal or defective hearing. Curves are then plotted showing the reciprocal of the minimum audible energy at the different pitches up to 5,000 d. v. Curves are also plotted for ears with deficient hearing showing at the deficient pitches how much more current through the receiver is required for the patients to hear than is required by a normal ear; such curves show the relative minimum audible current at the various pitches compared with the normal.

"Experimental Results.—Tests which have been made on normal ears—that is, ears which when examined by an otologist show no physiologic defect, show that the maxima of sensitivity occur at various pitches, but these maxima are not in the same position for all ears. There is also a wide difference in sensitivity among normal ears, but in general they

are all alike in that they are most sensitive from 300 d. v. to 5,000 d. v., the range in pitch which covers the regions most important for speech and music. By precise physical measurements we become amazed at the extreme sensitivity of normal ears compared with the extreme insensitiveness of abnormal ears; thus it may require a sound energy as much as 50,000,000 times above the normal pitch for the patient with nerve deficiencies to detect, say, a pitch of 4,000 d. v.

"A large number of curves have been taken on abnormal ears, and these curves have been correlated with the otologic findings. Internal ear deafness (nerve deafness) shows a depression in the higher frequency range (say 3,000 d. v. and above), without changing the sensitivity at the lower frequencies.

"Curves for middle ear deafness involve primarily the lower ranges and are characterized by a larger decrease in hearing for the lower tones, near 1,000 d. v., than for the higher tones, say above 3,000 d. v. The patients may have serious internal ear deafness, with little or no knowldege of it, but middle ear deafness, since it reduces the sensitivity for tones important for speech perception, makes one conscious of trouble as soon as the middle ear begins to be involved. In diseases involving the middle and internal ear it is difficult to say how much of the deafness is due to the middle ear and how much is due to the internal ear.

"Theoretical Bearings.—The theoretical bearing of the data so obtained has been discussed elsewhere. (Wilson and Minton: The Proceedings of the Chicago Institute of Medicine, pp. 157-171, 1921; Minton, John P., Proceedings of the National Academy of Arts and Sciences, September, 1921; Physical Review for 1922.) It is sufficient here to say that by correlating the data so obtained in a study of both normal and abnormal ears much light has been thrown on the manner in which the ear appears to function physically."

DISCUSSION.

MR. VERNE O. KNUDSEN, department of physics, University of Chicago, opened the discussion and said that a telephone receiver energized by a current from a vacuum tube oscillator is used as a source of sound for determining the sensibility of the ear to small differences of loudness and pitch. Tones varying in frequency from 30 d. v. to 20,000 d. v. are produced by the oscillator. By means of a divided resistance circuit the intensity of the tones can be varied by any desirable and measurable intervals from the threshold values up to very high values.

Some auxiliary experiments showed that the acoustic energy developed by the receiver diaphragm is directly proportional to the electrical energy which actuates it. The electrical energy is therefore a convenient measure of the relative acoustic energy developed by the receiver diaphragm.

Data that have been taken thus far on a limited number of ears show that the sensibility of the ear to small differences of intensity, measured by the ratio of the smallest perceptible increment in energy to the total energy, AE/E, is—

1. Dependent upon the intensity. The ratio AE/E decreases as the intensity increases. For a wide range of moderate and high frequencies the sensibility is nearly constant. Its value for ordinary frequencies is roughly 0.10.
2. Nearly independent of the frequency between 100 d. v. and 4,000 d. v.
3. Nearly the same for all normal ears.

The sensibility of the ear to small differences of frequency, measured by the ratio of the smallest perceptible increment of frequency to the whole frequency, is—

1. Dependent upon the intensity.
2. Dependent upon the frequency. The sensibility ratio decreases from about 0.01 at 50 d. v. to about 0.002 at 1,000 d. v. (for a single ear). For higher frequencies the ratio increases.
3. Dependent upon the mode of reception. Binaural reception yields a resolving power which is about twice as high as the resolving power of a single ear.
4. Only approximately the same for different individuals.

From these data it may be inferred that the average normal ear can distinguish about 400 graduates of loudness (for pure tones of moderate loudness), and over a frequency range of ten octaves (20 d. v. to 20,000) the average pair of ears can distinguish about 5,000 musical intervals or gradations of pitch.

Dr. J. Holinger asked how the flexible tubes influenced the transmissibility of the sound in the hearing device, and whether the smoothness or roughness of the inside of the tube had any influence.

Dr. Sabine, replying to Dr. Holinger, said that the tests as he made them did not bear directly on that point. Theoretically the fact that the tube is flexible would tend to reduce somewhat the intensity of the sound which it transmitted, but that would be a slight reduction, in the order of 50 or 100 per cent, whereas the multiplication would be two or perhaps three hundred fold, so the flexibility of the speaking tube was not an objection to it. Theoretically the smoothness or roughness would have some influence but this effect would be relatively small.

Dr. Alfred Lewy recalled one case which was a stapes ankylosis which presented some very obscure labyrinthine symptoms. The labyrinthine symptoms were not clear cut, and neither were the tests of the hearing clear enough to be certain of the diagnosis. Mr. Minton's diagnosis was spongification of the capsule, and he wondered whether this had not affected the labyrinth sufficiently to interfere with the function of the vestibular nerve. So far as they could tell this was what happened.

Dr. Lewy thought the greatest practical value of Mr. Minton's work rested in the check that could be maintained on the improvement under treatment. The ordinary tests were not sufficiently delicate to determine whether the patients are being helped or not, and he considered this work of great practical value in this way.

In Mr. Knudsen's work, his findings with his much more accurate instrument corresponded almost exactly with Dr. Lewy's experience with the monochord. He had one man, a trained musician, who seemed to hear up to 22,000 He was going to ask Mr. Minton to see this patient and ascertain if he really heard that high.

Dr. Sabine said that in cases of incipient otosclerosis it had occurred to him that a remedial effect might be obtained by stimulation of the ear by intense sounds. For example, in cases where there was some deafness he thought one might stop the progress of the defect by exercise with sounds, and asked the opinion of the members as to whether there was any rational basis for this belief.

Dr. Sonnenschein said that in the minds of the general practitioners, not the trained otologists, the idea of otosclerosis was that of an actual sclerosis, but this was a misnomer. The condition is really a spongification of the bone and its replacement by a very vascular bone, which interferes with the movement of the stapes. Many methods and devices have been used without avail to overcome this condition, and he wished someone would discuss the effect in those cases where a great deal of vascularization is already present

Dr. Joseph C. Beck was very much interested in the presentation of the subject. The point about the pathologic change in otosclerosis and other conditions around the footplate of the stapes seemed to be not pertinent to the otosclerosis but to other changes of inflammatory character whereby the stimulation by sound would be of value. He thought all would agree that the difficulty with many people possibly is that very early, when they hear but do not understand, they get into the habit of not listening, and so by stimulating them, by insisting upon their listening to tuning forks both by bone and air conduction, this would be of considerable value in the absorption of the inflammatory by-products.

Dr. Beck also called attention to the commercial houses that grasp such scientific devices as this, and that there is an oscillator for therapeutic purposes making a buzzing sound for producing hyperemia and absorption. In the East this appliance is being used largely, and some of the good men in that locality claim that they get results. A good deal had been mentioned about the oscillator at the last meeting of the Academy, and since then he had received a number of letters asking where such a device could be obtained. It seems that people generally are after some device for improving the hearing. In his practice he recommends devices, particularly those of an electrical nature, and thought that people get a great deal of benefit from them. Perhaps this is true only in one small area, as borne out by the charts, but he believed the use of devices for stimulating and improving the hearing, such as the phonograph and other means, were of great benefit. In the otosclerosis, in which we recognize the spongification and encroachment, this stimulation would not be in place, but in other inflammatory conditions he believed it would be of value.

DR. A. A. HAYDEN said he had been intensely interested in the papers and in the subject because of the fact that at least two members of his own family, his mother and an aunt, have otosclerosis. It has been his privilege to view rather intimately the feelings with which these people take up artificial means of hearing. While his mother can hear very well with an electric hearing device, and while many of his patients are greatly aided by these electrical devices as well as by other aids to hearing, speaking tubes, etc., he finds great reluctance on the part of nearly all these people to make use of these devices on account of the rather conspicuous appearance. He did not know how many cases of otosclerosis he sees in a year but thought it was the exceptional case who uses a device and the exceptional individual with nerve deafness who uses a speaking tube. The very first speaking tube he ever was instrumental in having a patient buy was just two weeks ago. It appeared that the people who are hard of hearing would rather keep their defect than use the external devices. This is a strange but true fact.

As to Dr. Holinger's remarks regarding spongification in deafmutes, he was interested to know if that was not a coincidence and not the underlying pathology. It would be perfectly possible for a mute to have a spongification engrafted upon the essential cause for his deafness, just as this evening it might me that the patient shown by Dr. Boot had something other than the cause suggested by Dr. Boot.

In regard to the exercise of the function of hearing with the idea of improving it, Dr. Hayden thought there were a number of facts that militated against it. The presence of boilermaker's disease must not be forgotten, and it must be remembered that that was the result of excessive noise. However, that was not a case in point. He did not believe that the use of the cochlea could increase its efficiency any more than the use of the retina with a hemorrhage would increase the individual's perception of light in that portion of the retina. It is exactly like looking for relief from paralysis in the eye from a muscle that does not functionate. He looked with the utmost skepticism for relief of loss of hearing from exercising with sound.

DR. MINTON stated that in certain patients who had middle and internal ear deafness he had noticed that stimulation with sounds at certain pitches had brought about sufficient temporary improvement to enable the patient to detect a sound about one-fourth as intense as before. They might require a relative receiver current of perhaps 1,000 initially, but while testing the patient this value may decrease to perhaps 500. This improvement is but temporary, for when the patient returns for testing the next time, the original value—e. g., 1,000—is obtained. It is not likely that we can obtain any permanent improvement in hearing by this means.

DR. PAUL E. SABINE thought Dr. Shambaugh had made the statement that in cases of nerve deafness the speaking tube might prove helpful, whereas in cases of middle ear defect the electrical device would be helpful. The physics of that puzzled him, but he noticed that other men referred to the same thing and wished to know if it was a well authenticated fact.

DR. HOLINGER said that he knew of many instances of middle ear deafness where the patient would not part with the electrical apparatus for a million dollars, whereas an elderly gentleman with nerve deafness had tried many of them and thrown them away.

He drew attention to the interesting cases of diplacusis, where the patient perceived a certain sound in different pitch with the two ears. He tested an organ player who had to give up his profession on account of diplacusis, which rendered his playing to himself and the community painfully dissonant. Dr. Holinger tried to produce consonants by varying the pitch of the forks before both ears, and found a variance in pitch of two to six tones in the two ears.

DR. SABINE thought this might be due to the fact that the maximum sensitiveness of the ear occurs normally for those tones for which the telephone is most effective, and for that reason the patient with nerve deafness would not be benefited by the large amplifications of these tones.

In one patient he had found the defect mentioned by Dr. Holinger and, since the deafness came on, even though she could hear music, it was very unsatisfactory. There was apparently a difference in pitch of the same tone when heard in the two ears. There might be a possible explanation if a difference in intensity is ever interpreted as a difference in pitch.

Mr. Verne Knudsen stated that one of the students at the university had told him that the sustained tones of the violins at the Tivoli were heard differently by the two ears. Examination in the laboratory proved that the same tones were heard a semitone higher by his right ear than by his left ear. His left ear was probably the affected one, since his pitch identification with his right ear agreed with normal ear identification.

Two or three weeks later he noticed that the effect had disappeared, and five months later laboratory tests showed that he heard normally with either ear.

Dr. Minton thought the explanation of the difference possibly was because the two ears may possess quite different sensitivities, and if the tones are not absolutely pure a patient may hear a certain tone with the good ear and quite a different one with the poor ear, since, near the threshold of audition the sense of pitch vanishes and it is impossible to make accurate judgment of pitch difference near this limit. For tones whose intensity is much above the threshold value it is absolutely necessary first to adjust the intensity so that the tones are heard with equal loudness when listening with one ear and then with the other one. When this adjustment is made one can then test to see if a tone sounds of the same pitch in both ears. These requisites make it practically impossible for the otologists to secure any reliable data on the so-called phenomenon of diplacusis.

Dr. Geo. W. Boot thought that the very fact that diplacusis occurs was one of the best evidences that there is a physical resonator in the ear. If the portion of the resonator, for instance, that responds to C^2 normally, gets weighted by some exudate, it no longer responds to C^2 but to some tone lower in pitch. This explains the situation that occurs in diplacusis, for the lower pitch, say C^1, causes the resonator on one side that normally responds to C^1 to functionate, and on the other side the resonator that normally responds to C^2, so that the tone C^1 is heard as C^1 and C^2 at the same time.

With regard to exercising the hearing in otosclerosis to cause the return of normal function, Dr. Boot was reminded of a horse that he once had that was the victim of a bone spavin. The pathology was slightly different, but there was exostosis present in the horse's joint as much as there is around the footplate of the stapes in otosclerosis. After exercising the horse for a short time the lameness would wear off and the horse would seem better, but the next time the horse was exercised he was lamer than before. It was just so with the ear in otosclerosis. If the ear was massaged there was improvement for a half hour and then the condition was worse than before. Not long ago he had a patient in an advanced stage of otosclerosis. She received a blow on the head and at once her hearing became very much better, to the temporary discredit of the doctor who had given an unfavorable prognosis, but in less than two weeks her hearing was as bad as ever.

With regard to hearing instruments, Dr. Boot believed patients with nerve deafness were not apt to be much benefited by the electrical instruments. Patients with fixation of the stapes and increased bone conduction were apt to hear better, for the instrument rests firmly against the side of the head and the sound waves can get in by bone conduction.

Dr. Minton was of the opinion that the mechanical theories of the phenomena of audition are wholly unsatisfactory and are rejected by the physicists, who based their conclusions upon precise physical measurements. Otologists usually believe that deaf people hear with a telephone against the ear because of bone conduction. There is no physical basis for such a belief. Numerous tests have been made to detect "bone conduction" when a receiver is placed on the ear. Negative results are always obtained. The real explanation is that the telephone, when placed over the ear, radiates all its sound energy directly into the auditory meatus and little or none of the sound energy is lost to the surrounding space.

Dr. J. Gordon Wilson said that during the war he observed a great many men made deaf by shell concussion (explosion). A number of these men were improved by stimulating the auditory tract by pure tones conducted through the mastoid bone if very deaf, or through the air if not so deaf. They improved to a considerable degree, but not entirely, and kept this improvement. This subject was discussed in a lecture before the Harvey Society in New York in 1917 and published in their series of lectures.

Dr. Sonnenschein emphasized the fact that it is very important for otologists to know more about acoustics and the diagnosis of such symptoms coming within their sphere. He thanked the gentlemen for coming and enlightening the society. He thought one of the most practical points made was the fact that the old time-honored "garden variety" of amplifying the sound by placing the hand behind the ear seemed to give as much relief as the modern metallic devices.

Another interesting point was with reference to the small amount of energy required to produce audition, as shown by a 40 watt light having enough energy to activate a string of receivers going about 25,000 times around the earth.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday Evening, January 9, 1922.

THE PRESIDENT, DR. ROBERT SONNENSCHEIN, IN THE CHAIR.

Pulsating Sphenoid.

DR. CHARLES H. LONG presented a case of pulsating sphenoid and said his reasons for presenting this patient were twofold. First, he discovered that he had escaped a bad result from the treatment; second, he discovered that the man had a pulsating sphenoid. The patient was a man of 59 years, who was referred to him because of deafness and tinnitus in the right ear. Examination revealed an ethmoiditis with a mucopurulent discharge down the middle turbinate. About the 19th of December he exenterated the ethmoid, and along the anterior third of the turbinate was a mass of granulations toward the frontal sinus. He opened the frontal cells, and when the middle turbinate was removed with the ethmoid cells he noticed a discharge from the sphenoid. He wiped this out and there was a severe reaction. The man had a badly developed septum from a former traumatism. The following day the right eye was projected and swollen, red and congested. He spent several sleepless nights following the operation and had considerable pain, not only in the back of the head but through the eye. The ordinary treatment was used—washing out the nose with saline and using a hot water bag to the eye. About a week after the operation he syringed the sphenoid and the reaction from this put the patient back to bed. He reported at the office two days later, and it was then that he had a pulsating sphenoid.

The cause of the pulsation was not altogether clear, and it was not clear why he got projection of the eye and cellulitis, unless the membrane between the orbit and nose was perforated, or else in the sphenoid there was no bony membrane between that and the internal carotid, or else there was necrosis of the external portion of the sphenoid and possibly some pulsation of the brain or a tumor. There was no pain and the hearing in the left ear was normal. In the right ear he could hear an ordinary conversational voice at ten feet and the whispered voice at four feet.

Dr. G. W. Boot asked if the carotid was bare in the cavity.

Dr. Long replied that he did not know. After having wiped the cavity out he was satisfied to leave it alone.

Dr. Edward P. Norcross presented a paper entitled:

"A Case of Acute Mastoiditis with Unusual Complications."*

*See page 501.

DISCUSSION.

Dr. Joseph C. Beck recalled a case of an abscess occurring in the side opposite to the mastoid operation. The patient gave absolutely no symptoms referable to a temporosphenoidal abscess in the side opposite the operated side, but died suddenly a week after the mastoid operation. Postmortem examination showed that it was an old abscess. The lining of the abscess cavity was very thick, and he thought probably the operation activated this old abscess and caused a direct extension into the region of the fourth ventricle.

Dr. G. W. Boot thought a good deal of light might be obtained by means of a lumbar puncture, and asked what the lumbar puncture in this case showed.

Dr. Otto Stein considered it a rather unusual complication and thought there were very few cases reported. When Dr. Dean reported his operation for a retropharyngeal abscess by the external method before the society, in discussing the operation Dr. Stein reported a case under his care similar to the one just reported by Dr. Norcross. He believed such cases occasionally occurred and were probably not recognized, but thought Dr. Norcross' case was a rather rare type of retropharyngeal abscess.

Dr. Robert Sonnenschein reported a case seen a month and a half ago, in an eight months old infant that developed a right sided otitis media. The right drum membrane was incised and profuse discharge escaped. Shortly afterward a retrotonsillar abscess developed on the right side. After this was incised a similar abscess appeared on the left, which also was opened, and following this another reappeared on the right side. Then the right cervical lymph glands, which had been swollen during the whole illness, broke down, were incised and the patient recovered. Streptococcus hemolyticus was found in the pus contained in the glands.

Dr. Norcross (closing the discussion) said that the spinal fluid was under slightly increased pressure. The cell count was 10 and the various tests were all negative. Roentgen examination of the mastoid, spine and skull showed nothing abnormal.

Tooth in the Right Bronchus.

Dr. Harry L. Pollock presented a young lady with a history of having a tooth in the right bronchus.

On June 30, 1921, this patient had a tonsillectomy performed in a small city in Ohio. The operation was performed under general anesthesia, with the patient's head in the operator's lap. The open method of anesthesia was employed first, then the blowing method, suction being employed. Just before the operation the patient warned the doctor that she had a left cuspid tooth on a pivot, and immediately after the operation she discovered that the tooth was missing. She called attention to this fact, and the physician told her that the operating room had been cleaned and the tooth had probably been swept up and thrown away. She recovered from the tonsillectomy within a few days and went to her home in Minnesota, and there developed a right sided pneumonia. She recovered from this, but still had symptoms of cough and pain in the chest. She was then taken to another hospital and told that she had tuberculosis and was treated for this condition. She continued to have symptoms of tuberculosis, but they were unable to find any bacilli. She was then sent to St. Paul, and for the first time a radiogram was made, which revealed the tooth in the right bronchus.

She then came to Chicago and was seen by Drs. Pollock and Beck, this being about five months after the tooth became imbedded in the bronchus, and Dr. Edwin McGinnis was called in to remove the tooth. The patient was put under Gwathmey synergistic anesthesia, consisting of three hypodermic injections of ⅛ grain morphin in two cc. of a 25 per cent magnesium sulphate solution, one-half hour apart. Following the last hypodermic three ounces of ether, with three drams of paraldehyde in three ounces of olive oil, was introduced into the rectum. The magnesium has a synergistic effect on the morphin and increases its effect greatly without affecting the danger of the morphin. In these cases it is almost impossible to give a general anesthesia on account of the infection and the pus. The radiogram showed a distinct abscess in the lung. Dr. McGinnis passed a bronchoscope and found the abscess cavity, but could not reach the tooth. It was then decided to let some of the inflammation subside and then attempt it again, but on the following day the patient coughed the tooth out. She developed a little pleurisy on the right side and a little more infiltration but had now completely recovered. The operation was performed three weeks previously and they were sure that had the capsule not been opened the tooth would not have been expelled.

This case was presented to emphasize the dangers of tonsillectomy and the fact that this patient had gone for five and a half months with a history of having lost a tooth, with pneumonia and socalled tuberculosis, before the radiogram was taken which disclosed the tooth in the bronchus.

Primary Lateral Sinus Thrombosis of Jugular Without Apparent Otitis Media.

Dr. H. Pollock presented a boy who was first seen on March 10, 1921. He had then had a cold and pain in the left ear for about a week. A moderate degree of otitis media was present. The child was very unruly. The mother was advised to keep him in the hospital, but he would not stay. He was reported to be better the following day, but two days later the pain was worse and he was again brought to the hospital. Upon the second examination the ear was still red, there was no bulging of the membrane, and hearing was poor. The temperature was 100 to 101 degrees F. He was kept in the hospital and hot compresses were applied. The second day afterward a thick, hard, painful swelling was noticed down the neck, and a diagnosis of lateral sinus thrombosis, with extension to the jugular, was made. The following day the sinus was opened, the thrombus removed and the jugular was ligated. The boy made an uneventful recovery. There was never a perforation of the ear drum nor any discharge from the ear. The ear was red but there was never any bulging. There was very little pus in the antrum, but it went through the middle ear into the lateral sinus. The boy's hearing at time of presentation was perfect.

In this case the Beck-Crowe test, made by compressing the opposite jugular, gave a positive result. There was great dilatation of the blood vessels on both sides. There were no symptoms of meningitis. The jugular was not dissected, but simply ligated down low. A small abscess formed, which was drained two days later. The wound was left wide open and permitted to granulate in.

The morning of the operation the temperature ran up to 102 to 104 degrees F. As soon as the operation was finished, although the bed was prepared with hot bottles and blankets, the boy had a terrific chill just as he came out of the anesthesia. The temperature never went up following this, but remained practically normal the rest of the time.

Atrophic Rhinitis.

Dr. H. Pollock said that there was nothing unusual in the diagnosis of this case. The disease had lasted for several years. Dr. Beck had performed a bilateral ethmoid exenteration. Following recovery from this she decided to go further with the treatment and have the place left by the exenteration fill in. They always do the ethmoidectomy by the Mosher method, with the middle turbinate intact. There was pronounced atrophy on both sides. They then implanted into the septum another septum from a previously operated case.

In this work they had carried out a series of experiments previously in trying to fill in this space, starting with paraffin. This proved unsatisfactory in most cases, and they then tried other substances. The first thing tried out was the implantation of fascia lata. This was satisfactory except that it was so small. In the first case they took both fascia latas and implanted them on one side, but they shrunk to almost nothing. Following this they tried fascia from another individual, but had very little success with this method. Most of it sloughed out. They then tried another method of doing a submucous operation on one side and then implanting the substance between the bony and cartilaginous septum. It does no good to put the implant up so high, because the atrophic condition is down low. They next tried the plug that has been used in osteomalacia, consisting of paraffin, iodoform and wax. This makes a hard substance that has been used in osteomyelitis, but in their work it did not prove satisfactory. It all sloughed out. So they resorted to the implantation of another septum from a previously operated case. They have done this in probably thirty cases and only one or two have sloughed out. The rest have healed beautifully. At first they tried to do both sides at once, but found that it took away the nutrition from the septum to remove both flaps, a small perforation followed and bone and cartilage sloughed out, so they now do one side at a time.

This patient was operated on the right side about three months previously and immediately showed improvement. The odor disappeared entirely, but the other side became worse. They then operated on the left side, and at present there was bulging on both sides.

It is sometimes necessary to use two septums in this operation. On the right side they made an incision far to the back so they could make one in front and dissect up the perichondrium and implant a second septum if it proved necessary.

The result was lessening of the discharge and disappearance of the odor. It had been a year and a half since they operated the first case and with one or two exceptions they had no infection.

DISCUSSION.

Dr. H. R. Boettcher asked if they closed the initial incision.

Dr. Pollock replied that they used a little collodion and cotton and a Burnay split was then kept in for twenty-four hours. The fascia was not used for a few hours after removal, but in all these cases operated after the primary ethmoidectomy they waited until they got a submucous that they knew to be free from infection and then implanted it within five minutes.

Dr. John A. Cavanaugh stated that he had operated upon several cases, using the method advocated by Dr. Pollock, but his results were not as satisfactory.

Dr. Cavanaugh transplanted a large piece of cartilage and bone removed from another case. It grew in very well and the patient progressed for a time, but the secretions soon returned. About a year later the man developed a severe nasal hemorrhage and consulted another physician during Dr. Cavanaugh's absence from the city. This physician told the patient the operation was poorly done and in order to get relief he would have to undergo another septum operation.

Pulsating Artery in the Left Pharyngeal Wall.

DR. H. C. POLLOCK presented a woman with tinnitus in the right ear and right sided otitis media. Examination of the nose and throat showed marked pulsation behind the posterior pillar, with a pronounced bruit on the left side. He believed this was not an anomalous artery but an aneurysm on the left side with an otitis media on the left. The tinnitus was probably due to the otitis media.

Pituitary Dystrophy.

DR. JOSEPH C. BECK presented a child, boy, aged nine years, who was almost completely blind. There was a history of severe headaches, vomiting and all symptoms of brain tumor. The child was very fat and entirely unable to walk. He presented the typical Froelich syndrome of pituitary dystrophy. The blindness had developed rapidly, with a sudden disappearance of the pressure symptoms, nausea, vomiting and headache. The intellect was not impaired at all. The child answered questions freely and sang and recited in an almost precocious way. He could not walk, as stated above, because his feet seemed unable to bear his weight.

By means of X-ray and other findings, they made a diagnosis of pituitary tumor, which had broken down into the sphenoid sinus region without obstructing the breathing, and they decided to perform a subtemporal decompression to see what result would be obtained. Light perception was restored, but he was still unable to walk.

Dr. Beck asked for an expression of opinion as to whether they should now do an intranasal operation, or should they do nothing more.

In reply to a question as to why he did not use radium, Dr. Beck said he had not thought of trying it in this case. He believed ductless gland therapy would be of no avail and thought the only question was whether further operation should be attempted. A neurologist had expressed the opinion that there was nothing to be done and concurred in the diagnosis.

(Note—Since the above presentation before the Chicago Otological and Laryngolegical Society, Dr. Beck operated upon the boy by way of the cranial route and found no evidence of a tumor in the region of the hypophysis. The boy appeared to have very much distended lateral ventricles. He did very poorly after the operation and two weeks later died from symptoms of a low grade meningitis. Postmortem showed that this was true. It further showed that he had no changes in the sella turcica. He had, however, a marked internal hydrocephalus.)

Carcinoma of the Larynx; Laryngectomy.

Dr. Joseph C. Beck next presented a man with a history of a growth in the larynx which had persisted for several months. This was treated by a local specialist, but he was finally referred to a large private clinic, where a laryngectomy was performed and was subsequently rayed with large doses of radium emanations. There was a recurrence and he was given more large doses of radium emanations and was then told that was all that could be done. The patient was then referred to Drs. Beck and Pollock because of choking sensations, and Dr. Pollock did a hurried tracheotomy. Following this they fed him up for one week or ten days, until he was in condition for a laryngectomy.

One of the interesting features about the case was that the operation was done under what is termed the synergistic anesthesia of Gwathmey. Dr. Beck reported fifty cases operated upon under this synergistic anesthesia, and exhibited a chart of the cases. The operations were the ordinary ones of the nose and throat, as, for instance, tonsils, septum, turbinectomy and ethmoidal exenterations, as well as the borderline and major operations about the head and neck. He stated without hesitation that he had never tried anything in the way of an anesthetic that worked so beautifully as this does. He had performed plastic operations without any pain whatever. They had sometimes found it necessary to give some additional ether or gas, as Gwathmey had stated in his articles. He believed there was no danger of toxic effects from this method and that there were very few untoward effects of any kind. In one case there was some irritation of the rectum for a day or two, and in one case mild infectious abscesses at side of injection developed. They had not used this method in children, but for a general anesthesia, like a sinus operation, where the patient could be kept sufficiently conscious to be able to answer questions, blow the nose, etc., he had never seen anything to equal it. In the near future they were going to publish their report on the first one hundred cases, giving the original technic as well as their modifications. At this time they referred anyone interested to Gwathmey's articles, which appeared in the A. M. A. Journal, or to Dr. Bernheimer, who has had this work in charge.

In the patient presented the operation was done according to the Glück method. They found that the growth was entirely intralaryngeal, not extending to the outside at all, and he felt that there would be no recurrence. They had used no radium and would not as long as things remained in normal condition. The man had developed erysipelas in the course of the treatment, from which he completely recovered.

Another interesting thing about the case was the matter of feeding, which was done by an Ewald duodenal tube in the beginning, but the patient was now able to take ice cream and some other things without the tube.

Hyperesthetic Rhinitis.

Dr. J. C. Beck presented this patient because of the work which had been done by Dr. Arnold B. Kaufman in this disorder. The treatment of hyperesthetic rhinitis was considered practically hopeless accordng to old methods. He asked that the privilege of the floor be extended to Dr. Kaufman, so that he might speak of his treatment of these interesting and difficult cases.

Paraffinoma.

Dr. Harry L. Pollock presented a patient who had sustained a broken nose, which resulted in a "saddle nose." A surgeon had endeavored to remedy this condition by injecting paraffin and the patient now had a real paraffinoma. The nose was red and in worse condition than before this operation. The skin was infiltrated and the growth was adherent to the tissues. They intended to remove the entire mass, right down to the nasal bone, and do a plastic operation, as these tumors frequently become malignant.

DISCUSSION.

DR. A. B. KAUFFMAN said the case presented was one of a series of types in which during the past year they had attempted to determine what part, if any, deficiency disease may play in producing any pathologic state or malfunctioning of parts in the ear, nose and throat other than fertile soil for the growth of saprophytes or pathogenic organisms. In animals it has long been recognized that the snuffles of hogs is primarily a nutritional disorder, and Hess has long called attention to the frequent coincidence of nasal diphtheria and latent or subacute scurvy. They made an effort to observe the possible relationship of dietary deficiencies, particularly vitamin deficiencies, to various nasal and aural conditions which were of more or less unknown etiology and which pathologically showed changes not of an inflammatory nature nor directly due to bacterial invasion. Of these, hyperplastic ethmoiditis and otosclerosis appeared to be the most conspicuous.

In hyperplastic ethmoiditis, histologically, they found the osseous changes were those of absorption, the bone cystic or thickened by a rarefying process. These pathologic changes are not unlike those found in other well defined deficiency diseases, so on this hypothetical basis they have given to one group of typical cases a dietary rich in water soluble B and C vitamins, while in the second group there was simply added to their dietary cod liver oil, which is abundant in the fat soluble A vitamin. The first group showed no particular improvement, while the second group began to show definite amelioration of symptoms. Therefore, on the basis that in this condition there may be a deficiency in the fat soluble vitamin, they had determined the inorganic phosphorus content of the blood because of the fact that investigators at Johns Hopkins Hospital gave sufficient basis to assume that the level of blood phosphates is determined in part by the amount of fat soluble A vitamin available for the body needs, and their results have shown for the most part a diminution in the blood phosphate content.

In otosclerosis they had more basis for the assumption that the condition may be a manifestation of a deficiency disease because of the analogy of the pathology in rickets, and in this condition it must be borne in mind that in rickets, although a condition of early infancy and childhood affecting young bones, there are definite changes in the temporal bone. Mayer in his recent book on otosclerosis studied the changes in the temporal bone in a nine months old infant with well advanced rickets. He found a definite bony degeneration in the cartilage about the oval window, and that the newly formed bony tissue in the region of the oval window remained uncalcified, the defect being a lack of calcification rather than the formation of cells.

Here, then, in rickets, a disease of infancy and childhood, and otosclerosis, a condition existing between the twentieth and fiftieth year of life, changes occur in the temporal bone that are not dissimilar, although their end-results are not quite identical. Furthermore, it is known that rickets is essentially a dietetic disorder, centered on the rôle of vitamins and more specifically on the fat soluble A vitamin; that it can be produced experimentally by diets deficient in this vitamin, and has been cured and prevented by the administration of cod liver oil, which contains the deficient accessory.

Can it not then be assumed as a working hypothesis that a similar deficiency, acting throughout a different period of life, may not produce such a clearcut disorder as in infantile rickets, but bony changes elsewhere? There are likewise certain blood findings in active rickets, the most important being a lowered content of inorganic phosphorus. This finding is considered as nearly conclusive evidence of active rickets. In some of these cases of progressive otosclerosis they have likewise found a diminution of the blood phosphate, and as mentioned before, this level is determined in part by the amount of fat soluble A vitamin available for the needs of the organism. They also hope to show changes in the temporal bone of animals which have been fed on diets deficient in the food accessories similar to those found in otosclerosis.

DR. CHARLES M. ROBERTSON said that he had contended that rickets and otosclerosis were practically the same thing. In Dr. Beck's case of pituitary pressure, Dr. Robertson thought he would be decidedly justified in operating upon the case, with the probability of finding a cyst rather than a tumor. The history of the case would seem to indicate that a cyst was forming, because of the rapidity of the increase in size. If a solid growth had formed with such rapidity the child would have been dead. If the growth was a cyst the prognosis was favorable as regards sight and life. Dr. Robertson had reported two cases of cysts of the pituitary, operated intranasally. In one case the vision was reduced to almost zero, and the optic nerve looked chalky, like a profound optic atrophy. When pressure was removed the vision came back to normal and the optic nerve assumed normal appearance. He thought to let the case go as at present meant nothing but disaster, and the child would be better off dead than in the present condition. He was in favor of operation on the chance that the growth was a cyst underneath the pituitary, and thought it would be crowded down so that it could be extirpated through the sphenoid sinus.

In the first case of carcinoma of the larynx, he thought the gentleman was very fortunate. He had a unilateral growth, which was taken out by the only method—from below upward. This is the easiest method of removing the larynx and the safest. One point in removal of the larynx is vital: The recurrent laryngeal nerve running up the other side toward the larynx is very easily irritated. Even if rubbed slightly it would irritate, and as the fibers of the pneumogastric are very intimately associated with the recurrent laryngeal nerve, respiratory paralysis may occur from irritation of these fibers. The fact that the patient had an erysipelas was in his favor. Dr. Robertson had seen two cases, one in the Royal Hospital of London and one case in private practice, where erysipelas cured such a growth. This fact was so well known that patients have been infected with erysipelas in order to promote cure. In one case seen in London the growth affected the larynx and the pharynx. When he examined the patient after having had erysipelas the contour of the larynx and the pharynx was as nearly normal as he ever expected to see them.

It was impossible to tell that the patient had had a tumor of malignant or any other type.

In his opinion about 40 per cent of the success in cases of laryngectomy depends upon the operator, and the other 60 per cent upon proper after-treatment. Patients die, not because they are not operated upon properly, but because they are in hospitals that are not taking care of such cases as a routine thing, thus prohibiting the proper care and after-treatment. This work needs a staff specially trained to take care of this type of case. They have to be kept clean, cleaned every half hour if necessary. The discharge that comes down from the throat is excessive, and if it gets into the tracheal wound there will be sloughing. He considered the operation of laryngeal fissure much easier. In the case presented, Dr. Robertson thought the man would recover a voice that would be husky, but one that would permit him to talk for hours, if necessary. He had always made it a practice to broil these cases with a hot iron after the diseased tissue was removed. Then the cicatrix forms a straight band across the larynx as a fibrous band.

Regarding the use of radium, Dr. Robertson expressed the opinion that it had killed ten times more people than it had even benefited, but what the future will develop cannot be told at present. He had seen many cases which radium had helped for a few weeks, and then the growth spread rapidly. In his service at the Alexian Brothers Hospital, where there were many cancers of the throat, he had yet to see any except the superficial ones that had been cured by radium or even alleviated. Outside of the extirpation of the tumor in toto, he believed there was only one thing to do: In his opinion, the application of actual heat was the thing that destroyed malignancy. It made no difference whether the growth was in the throat or some other part of the body, and if one used the knife he should sear the wound afterward. Doyen, in Paris, uses voltaization and fulguration, which char the tissues and they heal. In cauterization under ether great care must be used or an explosion of the ether gas may result. The first time he ever tried this he was afraid the patient would explode. After opening the trachea, he used the Paquelin cautery, and in order to avoid exploding the patient he waited until after he had expelled all the air in the lungs and then cauterized before the act of inspiration. He had since seen the cautery used without bad result, but warned of this danger.

Regarding Dr. Pollock's case of sinus thrombosis without rupture of the drum membrane, Dr. Robertson had seen two cases of sinus thrombosis in which there never was discharge in the ear, but at the time of operation the attic cells were full of pus. He did not know whether one was a tubercular case or not, but the drum was either resistant or there was not enough pus to break through by pressure. The suppuration had been going on until the patient was beginning to have profound meningitic symptoms. The eardrum membrane was not even pink, and yet it was an old case of cholesteatoma.

Dr. G. W. Boot said he had had several cases that showed peculiar lung symptoms. In one case the child had inhaled a toy arrowhead. Another patient had inhaled a collar button, which had remained in the lung for two years before a diagnosis was made and at that time an abscess had formed. A third case, a boy, of about fourteen years, had irregular symptoms of tuberculosis, and a radiogram showed a piece of wire in the lung. The symptoms dated from about the time of a tonsillectomy and the wire was probably from a tonsil snare.

Dr. Boot thought it strange that general practitioners were so slow about taking X-ray pictures in peculiar pneumonia cases.

In his opinion the three great contributory causes of carcinoma of the mouth and throat are a dirty mouth, syphilis and tobacco.

Dr. Harry Kahn thought that Dr. Kaufman's recent work was epochmaking. He knew the results obtained in some of the cases treated were remarkable. There had recently been discussion in the Journal of the A. M. A. as to rickets as a vitamin deficiency disease. Some English believe there is an antirachitic vitamin. The Americans do not take this so seriously, and Hess has stated that the A vitamin is not a necessary article of food in the treatment of rickets and recommended the application of sunlight in order to prevent rickets. In the same paper a table of percentage of blood phosphates, both in normal and rachitic children, is given. H. Steenbock

(Science 50, 352-1919) made an interesting generalization in regard to fat soluble A vitamin. All food containing fat soluble A vitamin is usually yellow in color—butter, carrots, sweet potatoes, yellow tomatoes, corn, etc., and should form a part of the diet.

DR. ROBERT SONNENSCHEIN asked whether it was not a fact that unboiled milk contained almost as much fat soluble A vitamin as most any other substance.

Secondly, whether it is not the organic iodin found in cod liver oil that is partly responsible for the good effects. If it were not for the iodin, would not unboiled milk alone be just as good as any other substance?

DR. ARNOLD B. KAUFFMAN, replying to the discussion, said that cod liver oil in otosclerosis is not new. As far back in literature as one could find reports, almost every otologist abroad had used cod liver oil in otosclerosis because of the good results that were obtained, and because of the similarity between otosclerosis and rickets. It was used empirically and no mention was made of the fat soluble A vitamin. As to the English and American viewpoint of cod liver oil in rickets, Hess had come out with sunlight and ultraviolet rays, and most authors admit that they obtain the same results as from cod liver oil, but get the effect from different angles.

In answer to Dr. Sonnenschein's questions, Dr. Kauffman said the cod liver oil contained the fat soluble A vitamin in abundance, particularly the unrefined oil. It is impossible to cure rickety infants with unboiled milk, but cod liver oil will cure them.

Dr. Kauffman cited the fact that when Col. Garrison spoke on gastrointestinal conditions in relation to deficiency disease recently, he stated that during his experience in the Himalayan mountains he had failed to observe anything that he could point out as due to deficiency. He had never seen a case of deafness during his service there. How much importance could be attached to this statement Dr. Kauffman did not know, but it seemed that in various climates where people live on a strictly vegetable diet and uncanned foodstuffs these conditions are apparently absent.

DR. JOSEPH C. BECK, closing the discussion, thanked Dr. Robertson for bringing out some of the points he had not taken time to emphasize, but which he considered important, but he took exception to his very emphatic statements regarding radium. He agreed that in many cases the results were not good, but felt that such emphatic statements from a man of Dr. Robertson's standing would put a damper on the use of radium before enough time had elapsed. In Dr. Beck's opinion, more people should use radium so that the results can be definitely determined. He had hoped to present a patient for whom they had performed a laryngectomy from within outward by the use of radium. The man had refused operation, but did not refuse the radium therapy. He considered radium a very important adjunct in the treatment of cancer, but at this time could report no cures.

"Conditions for Progress of Otolaryngology."

Dr. J. Holinger addressed the society on "Conditions for Progress in Oto-Laryngology."

During the last thirty years our knowledge of anatomy, physiology and pathology of the ear has been revolutionized in all its particulars. To these advances America has contributed very little. Dr. Shambaugh has done very creditable work in anatomy of the labyrinth, but besides that little can be shown. It is questionable whether Germany, Austria and Switzerland will be able to continue with this tedious and expensive work as they have done so far. It is time for America to take an active part. The writer has discussed the conditions for doing advanced work with his former chief, Prof. Siebenmann of Basel, Switzerland, and can formulate those conditions under three main heads.

1. A large ear, nose and throat clinic with 10,000 patients a year.
2. Extensive laboratory facilities.
3. A great deal of fresh postmortem material.

All this with a sufficient staff of well paid assistants and a competent leader who can work together for years in connection with graduate and postgraduate teaching.

Dr. George E. Shambaugh was very much pleased to hear Dr. Holinger's remarks, because they brought up subjects which deserve serious thought. Dr. Holinger pictured a situation in regard to the specialty of otolaryngology in Basel.

We have nothing in this country that parallels this situation, and it is not at all likely that our development will be exactly along the same line. We have our own problems which must be solved in our own way. Dr. Shambaugh pointed out that there were some deplorable practices in connection with the treatment of patients suffering from nose and throat conditions at present, which have come about largely from the practice of instructing interns in general medicine and general surgery in the technic of operations in the nose and throat. These men can have no proper appreciation of the indications for these operations, and the result is that we are seeing a great many unnecessary operations by this type of practitioner. In certain ways there has been as much substantial progress in our specialty in this country as there has been abroad, but what has been accomplished has been worked out largely through individual initiative. We have had no assistance in the way of departmental equipment or organization, which is essential in carrying out any sustained piece of research.

There was one phase of the subject which Dr. Holinger did not touch upon, but which is quite vital when considering progress in our specialty, and this is the method employed in training men for this work. In Europe the prospective specialist has always gone through a period in which he served as an apprentice or assistant in a well organized and equipped clinic. Our own men who have sought work abroad have very rarely been able to avail themselves of this plan, which is reserved for their own men. A system of intensive courses, which might well be termed courses in "forced feeding," have been developed to accommodate especially the Americans. Courses of this sort are of value chiefly for those who have had their period of training by serving as assistants in a well run clinic, but they have no proper place for those who are undertaking to learn the subject without that training. The result has been the flooding of this country with a great many men whose training has been a spurious preparation based upon facts ladled out to them in these courses. The real training is acquired only by men taking part in the actual work—that is, serving as clinical assistants. One learns very little by sitting around listening to others tell how they do things.

Dr. Shambaugh thought that the facilities for securing proper training in otolaryngology were certain to be greatly improved in the near future. The fundamental training must be gotten here in our own institutions. After this has been acquired, courses such as are offered in our postgraduate institutions, as well as in certain foreign cities, might well be taken, but such courses should not be offered as a substitute for the real fundamental training. Those of us who occupy positions in our teaching institutions should bear in mind the difference between the opportunities in our own universities and those that exist, for example, in Basel, and should strive to educate our universities to a proper appreciation of what a department in otolaryngology should be.

Dr. Elmer L. Kenyon said in regard to research being placed on a scientific basis, Dr. Shambaugh had discussed one phase. There were still other phases which might be discussed. In his opinion, Dr. Holinger had struck a keynote which was right, and he thought the speaker had struck it at the right time, for we are on the point of paying salaries to scientific men, even scientific medical men, and this will do much toward placing research on a truly scientific basis.

Dr. Kenyon desired to express two thoughts in the encouragement of Dr. Holinger's suggestion: first, he doubted whether in the history of this country anybody had gone to the State Legislature and said this thing should be done; but we know that where other men in other walks of life have gone to State Legislatures and said that certain things should be done they often have been done. In his opinion, what was needed was a body of men who could establish an organization to set the thing going. Secondly, we occasionally do build great things at once. The Rockefeller Foundation was all built over night, as it were. But as a rule we do not build in that way, but rather with small beginnings, followed by gradual accretions. He believed the thought should be to start things now in the right direction, but not necessarily to expect this generation to put the project on a final basis, but perhaps the next, or even the third generation. We should attempt to set something going that is solid, and look for future generations to build on it. In this way, he believed, the substantial research foundations were more likely to be built up, rather than through some great act fulfilled all at once.

Dr. G. W. Boot agreed with all the speakers and believed there was probably no one particular in which the members were so deficient as in the pathology and histology of the subject. Probably with the exception of Dr. Shambaugh, there were not half a dozen members of this society who had ever cut a section of a temporal bone. We talk very learnedly of otosclerosis being connected with rickets, as the result of focal infection, and the result of syphilis, but we know nothing definite about it. It is all supposition, and we will never know anything definite until something was started such as Dr. Holinger mentioned. If this is not started it will be the same old story—"the fault, dear Brutus, is not in our stars, but in ourselves that we are underlings."

Dr. Boot moved that a committee of six men, members of this society, be appointed to see what could be done in this connection, the committee to consist of the president, the secretary, Dr. Holinger and three others, the object being to get such a foundation started.

Dr. Joseph C. Beck seconded Dr. Boot's motion, and said all were familiar with the political side of the question. Politics played a great röle in the securing of postmortems. All had had experiences with the undertaker politicians who prevented postmortems. They often have to fight for them, even though the families of the patients are willing to have them performed. Dr. Beck has reported this many times. The sentiment is decidedly against postmortems in this country, sometimes because of religious sentiment—this particularly among the Jews, but in the old country it is almost a law that postmortems shall be performed.

Another point is the matter of pathologic investigation. He offered the necessary facilities for this training, but had found very few men who took the necessary interest. They did not see the importance of this side of the subject but were interested only in the operative side, and the men who came for postgraduate work cared nothing for pathology.

Dr. Beck thought an earnest effort should be made by the society to start the thing suggested, and even if it took a hundred years to accomplish it, they should get started.

Dr. Kenyon thought procedures should not be complicated any more than necessary.

Dr. Boot thereupon, with the permission of the seconder, withdrew his motion.

Dr. H. R. Boettcher agreed with Dr. Beck that it is almost impossible to obtain postmortem examinations.

Dr. Harry Kahn thought two factors entered into the question: First, better education of the medical students; second, the apathy and antagonism of the public toward the medical profession. Anything the medical profession brings forth will be combated by all the sects and "isms," and unless they have a large lobby they will not get anywhere. If they have nothing that interests the State Legislature they will get nothing from it. The public has to be educated before such things can be put through. In Europe everywhere the doctor is looked up to and respected and honored.

Dr. Holinger, closing the discussion, thanked the gentlemen for their remarks. He thought the conditions here and abroad were not essentially different. He had seen the whole clinic in Basel grow out of nothing. When he worked with Siebenmann in 1891 the clinic was held in the private consultation rooms.

In regard to the postmortems, he thought they had the same prejudices to fight over there as we have here.

He had not brought this matter up before but had had it in mind for many years. This much is certain, nothing can be accomplished unless we make a start. He believed if the public was educated to the questions that need to be solved it will become interested and will help with funds. Politics and other difficulties can be overcome if the profession will work together. He had brought the matter before the Council of the Chicago Medical Society and felt that when there was unanimity much would be accomplished.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

DR. HILLIARD WOOD, PRESIDENT.

February 20, 1922.

Fracture of the Skull.

DR. HERSCHEL EZELL presented Mr. E. R. K., aet 45, motorman, who gave the following history: Twelve years ago was hit in right temple with hammer, resulting in scar about 1½ inches long, located above and in front of right ear. January 4, 1922, was in street car collision and fell, striking head on register in front part of car. Was not rendered unconscious by blow, but felt dizzy and unsteady following accident. Thirty minutes later had expectoration of blood, with discharge of blood from right ear. Few days later aural discharge became purulent in character and continued so for about one week. Patient complained of deafness, pain, discharge and occasional tinnitus right ear since accident. Alleges that previous to injury the right ear was normal. No trouble alleged in left ear.

Examination showed right ear drum retracted, scarred and discolored; no perforation; no discharge. Watch and tuning forks not heard by either air or bone conduction. The stethoscope test was used in an effort to detect malingering, but no hearing could be demonstrated in right ear. Left ear normal; hearing perfect by both watch and tuning forks. Nose and throat negative. X-ray by Dr. H. S. Shoulders shows fracture 1½ inch long in temporal bone, slightly above and in front of right external auditory canal. It is believed the injury extends to the base, but this could not be demonstrated by X-ray plate.

In presenting this case, Dr. Ezell asked an opinion as to whether this fracture of the skull, as revealed by the X-ray plate, could be the result of the injury some twelve years ago or is the result of the more recent injury.

DISCUSSION.

Dr. W. G. Kennon suggested that Dr. Shoulders, who made the plate, could give more definite information on this point.

Dr. Hilliard Wood, referring to the stethoscope test, said that he had become familiar with this test in examining Army applicants during the war. It was his opinion that this test is of doubtful value unless the rubber tubes are some three or four feet long—i. e., long enough to enable the examiner to stand some distance behind the patient; otherwise the patient will hear with the good ear by air conduction even though the other ear is deaf. He regards the stethoscope test as especially valuable in detecting malingering. He said that another good test, similar to the laerm apparatus test, consisted in irrigating the good ear and talking to the patient during the irrigation, the patient being unable to hear with the good ear during the process of irrigation. Dr. Wood also pointed out that in this type of cases, sooner or later, the question of damages arises, and in the matter of final settlement the doctor is virtually an umpire, as upon his statement the final awards are usually made; so that one cannot be too careful in these cases, in order that full justice may be done, both to the injured person and to the company which pays. He suggested that a Barany test might give valuable information in this case.

Dr. M. M. Cullom regards the noise apparatus test as better than the stethoscope test in detecting malingering. Another valuable test mentioned by him is the closing of the good ear and requiring the patient to count; if deaf in the ear being tested, the voice will be raised.

Dr. Eugene Orr, speaking of the various tests, said that he considered no one test to be absolutely infallible, as his experience both in Army service during the war and with the War Risk Bureau since had been that the majority of the men are coached in advance and are perfectly able to pass any test which may be given. Regarding the fracture, he advised the use of the fluoroscope, as he believes it to be almost impossible to diagnose fracture of the skull accurately except by the fluoroscope. He thinks the failure of the X-ray lies in the fact that every point of the skull cannot be subjected to the maximum intensity of the ray.

Laryngitis.

Dr. E. B. Cayce presented Mr. H. F. B., aet 50, who first consulted him November 20, 1921, complaining of hoarseness, gradually increasing for the past four or five months. The larynx at that time showed posterior portion of arytenoids swollen and interarytenoid fold pale and congested. Left vocal cord showed two nodes, with tendency of right cord to overlap in phonation. Tuberculosis suspected. Sputum examination by Dr. Clinton E. Brush, negative. Patient went to Washington, D. C., where he delivered several addresses, after which throat cleared up entirely until recently. Several weeks ago same type of symptoms recurred. Wassermann negative.

Dr. Cayce said that the larynx was so suggestive of tuberculosis that he was very much surprised at receiving a negative report on the sputum. While he does not believe that a positive diagnosis can ever be made from mere inspection of the larynx, yet there are several elements in this case which are very suggestive of tuberculosis—i. e., the linea alba of the hard and soft palate, extending down to the tip of the uvula, this condition being regarded by Dr. J. A. Witherspoon as especially suggestive of tuberculosis; the interarytenoid condition, the arytenoids being swollen and rather faceted, and the fact that the arytenoid fold is not obliterated during phonation. Dr. Cayce asked for a diagnosis in this case.

DISCUSSION.

Dr. W. G. Kennon said that while he had never seen a case exactly similar to this, yet to him it did not present the appearance of tuberculosis, the condition being one of edema rather than infiltration. He suggested that it might have been caused by the excessive use of the voice while suffering from laryngitis.

Dr. Herschel Ezell also doubted that the condition was tuberculosis, but found nothing upon which to base a definite diagnosis. In the absence of positive evidence, he advised antiluetic treatment, for a negative Wassermann does not disprove lues.

Dr. Cayce, replying to Dr. Ezell, said that in the presence of a negative Wassermann and the history and findings failing to indicate luetic trouble, he saw no reason why anti-luetic treatment should prove beneficial.

Dr. M. M. Cullom said that his observation had been that tuberculous laryngitis does not come on with the acuteness with which this condition developed; primary laryngeal tuberculosis being almost unknown. Under these circumstances, and in the absence of any general symptoms of tuberculosis, he would hesitate to make a diagnosis of tuberculous laryngitis.

Dr. Hilliard Wood pointed out that tuberculous laryngitis seldom if ever occurs except in fairly well developed pulmonary tuberculosis. He does not recall that in his experience he has ever failed to find pulmonary tuberculosis where he had a case of tuberculous laryngitis. He suggested that this condition might be one of malignancy, the age of the patient rendering it entirely possible. He agreed with Dr. Ezell's suggestion that antiluetic treatment be instituted, as occasionally this works miracles, even in the presence of a negative Wassermann.

Radical Mastoid for Chronic Suppurative Otitis Media With Cholesteatoma.

Dr. Fred E. Hasty presented Mr. A. J., aet 25, who, for the past 17 years has had frequently recurring pain and discharge from each ear, following some unknown type of fever. Some ten years ago had tonsillectomy, without any improvement in the ear condition. Seven years ago had tonsillectomy, following which the ears cleared up for two or three years. During an attack of influenza three years ago both ears began discharging, the left somewhat more profusely than the right. Pain more or less constant on left side of head, more especially in the mastoid region. Several otologists were consulted; all recognized methods of treatment were tried, but with little or no success.

Patient first seen by Dr. Hasty in fall of 1920. Upon examination right ear showed profuse offensive discharge; membrana tympani thickened, red, with a perforation in the posterior inferior quadrant. Left ear showed profuse offensive discharge; membrana tympani practically destroyed; considerable granulation tissue and polypoid degeneration. Functional examination:

Right		Left
6″	Whisper	....
8′—10′	Conversation	....
....	Shout	1′ (?)
128	Lower tone limit	1024
2048+	Upper tone limit	2048
Increased	B. C.	Markedly increased
Weber to the left		

X-ray showed only sclerosis in both mastoids. Advised to have radical mastoid left ear. Patient was not seen again until Nov. 1, 1921, when the ears were found to be in the same condition as one year previous. Radical mastoid advised and done on left side, with following findings: Sclerosed cortex and cells down to the region of the labyrinthine capsule, where a cholesteatoma extending from the antrum posteriorly to the sinus wall and upwards to the fornix of the inner table of the middle fossa; ossicles, excepting the stapes, were practically destroyed. Postoperative history uneventful.

This case was demonstrated to present some of the problems of radical mastoid surgery, as follows:

1. Discharge from both ears.
2. Left ear practically deaf.
3. What will happen to the right ear if we do, or do not, operate on the left.
4. Extent of pathology in left ear. X-ray was not helpful.
5. Cosmetic results of operation.
6. Patient no longer has headache; has gained about five pounds in weight.

DISCUSSION.

Dr. Hilliard Wood congratulated Dr. Hasty upon the beautiful result obtained in this case. He inquired as to the size of the cholesteatoma.

Dr. Herschel Ezell was impressed with the fact that there was so little deformity resulting from the operation, and asked the secret of success in this respect.

Dr. Hasty, replying to Dr. Wood, said that the cholesteatoma extended from the antrum back to the sinus wall and upward towards the middle fossa. This was all cleaned out up to the smooth cortex on the floor of the middle fossa. In answer to Dr. Ezell's question, he said that the character of the flaps, both the internal and the plastic flaps, was chiefly responsible for the lack of deformity. He described in detail the Richards method by which this operation was done. He also emphasized the fact that the X-ray failed to show this cholesteatoma. In his opinion, in these old chronic cases the X-ray shows nothing except the anatomy.

New Instruments.

Dr. Fred E. Hasty exhibited two new instruments as follows:

ANTRUM IRRIGATOR.

The object of this instrument is to combine a probe and small antrum irrigator. I find it to be satisfactory in washing any antrum, but especially in those cases where the opening is small.

(D) A solid round tip.

(E) Opening on side instead of end.

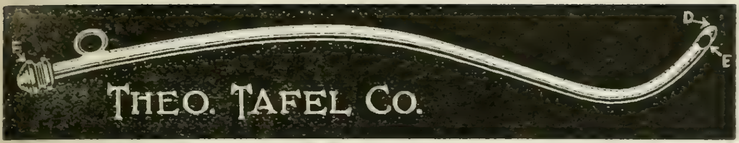
SUCTION ATTACHMENT ON TONGUE DEPRESSOR.

Since so much attention is being given to lung abscess following tonsillectomy, it behooves one to take every possible precaution in dealing with the blood, pus and secretions in the mouth during the operation.

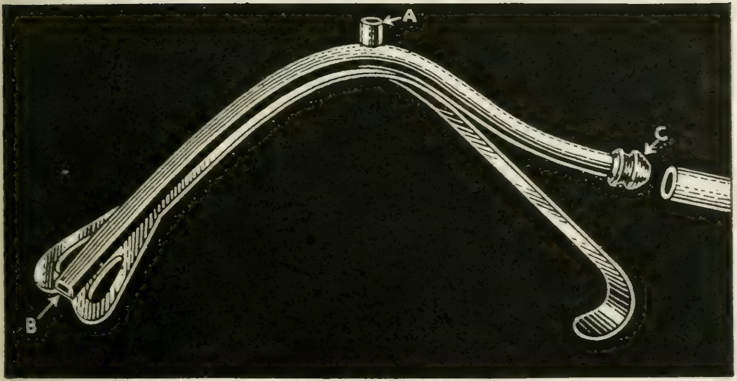
I have devised this suction attachment, which may be made on most any ordinary tongue depressor. Since this drawing was made I have had a tongue depressor made by shaping two pieces of metal together and leaving a groove between to take the place of the superimposed tube. This instrument has proven very convenient in my hands and does away with the necessity of the ordinary suction tube, and in that way it almost lends another hand to the operator.

(A) By covering this opening with the thumb, suction is produced at B on the end of the tongue depressor.

(C) For connection with rubber tube on any ordinary suction machine.



Antrum Irrigator.



Suction Attachment on Tongue Depressor.

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XXXVIII.

THE TREATMENT AND PROGNOSIS OF CARCINOMA OF THE LARYNX.*

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TOKYO.

The secretary of the American Laryngological Association has honored me with an invitation to speak before this illustrious assembly. I have decided to speak on "The Treatment and Prognosis of Carcinoma of the Larynx," a subject which I have specially studied for the last twenty years.

As you know, the history of the treatment of this particular disease is a long one and the literature on this subject is so extensive that I must forego entering into it. We know, however, from Sendziak's laborious literary studies that, already twenty-five years ago, four periods could be established with regard to the views on the treatment of laryngeal cancers: that of therapeutic nihilism, the birth of a rational therapy, and the period of wavering views having been passed through in succession, while at that time one could speak of a period of sober criticism. Since that time the therapy of laryngeal cancer has been further advanced on the one hand by a better diagnosis of its earliest forms and on the other by valuable and almost perfect modifications of the surgical methods, so that we may be justified in considering our present time that of achievement in the treatment of this malignant disease. Thus Felix Semon (London) was able to say in a lecture, which was held not many years before his death, before the Japanese Medical Congress, that carcinoma of the larynx, if diagnosed in its early stages, was curable in 80 per cent of the cases by laryngofissure and excision of the soft parts, a method which your countryman, Solis Cohen, has applied with very good results. Also Gluck (Berlin) is said to have obtained excellent results, even in rather advanced cases, by his applied modification of Billroth's total extirpation, first experimentally tried and later practically in over 300 cases. In a lecture given at Vienna in 1913 he was able to state that in a series of 86 operations he had only one case ending fatally, while in another series of 75 not a single patient died. The results of these two renowned surgeons seem to leave nothing to add to the surgical therapy of laryngeal carcinoma by giving our experiences and studies. But other writers give less favorable reports. Especially the Iitalian authors Gradenigo, Egidi and others would prefer total extirpation of the larynx instead of the thyrotomy recommended so warmly by Semon and Chiari. As to the total extirpation of the larynx, the results of different surgeons have been various. Many, though following closely Gluck's method, have obtained less favorable results, which was already confirmed by the reports of N. H. Pierce, W. Freudenthal and others here in America. Moreover, it seems to me that general surgeons who take an interest in cases of laryngeal carcinoma but being less acquainted with the smaller special laryngeal operations, as, for instance, with the endolaryngeal, are too much inclined to resort at once to the great total operation, even in early cases. On the other hand, laryngologists often delay the total extirpation, notwithstanding proper indications for it. For these reasons I do not deem it superfluous to take up this question once more, though Otto Chiari thirteen years ago, at the annual general meeting of the American Laryngological, Rhinological and Otological Society in Atlantic City, spoke on the same subject in detail. During more than twenty years of academic and practical activity I have employed various methods in operating on 141 cases of laryngeal carcinoma, and the results obtained and the facts observed have led me to views differing somewhat from those of the above mentioned authors.

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*Read before the annual congress of the American Laryngological Association, May, 1922.

First of all, I will speak on the surgical methods which I used and the results obtained, following up with a comparative criticism of other methods taken from the literature on the subject, and ending up with some general conclusions on the subject of laryngeal carcinoma.

The methods employed by me were five in number, viz.: (1) Endolaryngeal excision, (2) thyrotomy and excision of soft parts, (3) partial extirpation, (4) semilateral extirpation, and (5) total extirpation of the larynx.

(1) THE ENDOLARYNGEAL OPERATION.

This method was first employed by B. Fränkel with good results on laryngeal cancer, but on account of uncertain results was rejected by most surgeons. Felix Semon was of opinion that early forms of laryngeal cancer, which according to Fränkel may be treated by an endolaryngeal operation, could be better and more safely treated with laryngofissure, and Gradenigo, Otto Chiari, Ch. Jackson, etc., also positively rejected this endolaryngeal method for the surgical treatment of cancer. Gradenigo was even of opinion that the cases of successful operation had not been cancerous at all and that the time of postobservation had been too short. Being a pupil of Fränkel, I am still of opinion that the earlier a beginning laryngeal cancer is diagnosed, and the more certain the diagnosis is as regards the purely local form of the affection, the more certain this small operation may be successful. This will not only preserve the physiologic functions of the organ and avoid a deformed outward appearance, but will also add to the fame of the surgeon as to his surgical dexterity. And if a properly diagnosed very small carcinoma happens to be quite local, especially on the free edge of the vocal ligament, the endolaryngeal operation can be resorted to with good prospects. I performed it 23 years ago for the first time on a patient from Kumamoto, 56 years of age, who on the free edge of the right cord had a ragged polypoid cancroid, the size of the tip of the little finger, which had not yet affected the ventricular membrane. The diagnosis was verified by microscopic examination of a testpiece and I accordingly performed the endolaryngeal operation in two sittings within two weeks, with such good result that the patient could be discharged from the hospital after two months. One and one-half years later I presented this man at the medical congress in Tokyo, together with the microscopic slides made before the operation. At this time the right cord showed granular regeneration and produced a good voice. Since that time I have performed the same operation on 15 patients, in whom careful examination showed only strictly localized polypoid forms of cordal cancer, not yet spreading to the ventriculum, and also on two cases with pedunculated ulcerous forms of larynx carcinoma on the free edge of the epiglottis. The former were mostly performed under the laryngoscope with the aid of the sharp larynx forceps, or double curette; in only three cases "Schwebelaryngoscopie" was applied. The pedunculated forms were operated under the laryngoscope with the galvano-caustic loop, as shown by Loeb in his work on surgery, and the cancers at the end of the epiglottis also with the aid of the laryngoscope and the double curette. The results were on the whole very good. In five cases repeated examinations showed a complete cure, viz. the first patient, who died 18 years later at the age of 75 of influenza pneumonia, was during all these years well and showed no signs of recurrence. The second case with right sided cord epithelioma remained well for 11 years, the third with left sided cord cancroid for eight years, the fourth with pedunculated carcinoma on the left aryepiglottic fold and the fifth with right cord epithelioma five years later was quite healthy and free of returnation. The other 11 endolaryngeal operations showed a relative cure; the patients left the hospital almost well, the scar was healed and no local relapse or glandular metastasis was visible. Half of them, which could be kept under observation, were well after more than a year. They were all exhibited at the monthly meetings of the Japanese Otolaryngological Society. Only in three cases, the results were not good; one case with medullary carcinoma of the left cord, which besides the local symptoms of a sharply outlined superficial and ragged polypoid growth on the middle of the left cord already showed a slight affection in motility of the left cord, had to be treated a second time endolaryngeally a month later on account of recurrence and, two weeks later, total extirpation of the larynx became necessary. The other two cases, one of carcinoma pendula on the tuberculum epiglottica and another of right cord cancroid soon showed local recurrence, but further treatment was refused.

With the above results of my endolaryngeal operations I have been well satisfied, but unfortunately in the majority of cases of laryngeal carcinoma the operation can not be resorted to, because when the patients come to us it is already too late. I must emphasize that the indication for this minor operation must strictly be that the tumor is sharply defined in a restricted place. In every case of such nature I am not satisfied with the ordinary laryngoscopic examination, but I try to examine the ventricular space with my ventricular laryngoscope in order to see if the ventricular arch is already affected or not, and besides any possible affection of motility of the vocal ligament with the aid of the mirror. These two points I consider of the utmost importance to ensure the possibility of a successful endolaryngeal operation. The affection of the membrana ventricularis is a sign that the carcinoma is no longer limited to the edge of the ligament, and a diminished motility of the cord, which according to Felix Semon is a very early symptom of laryngeal cancer, is in my opinion proof that the cancerous infiltration is no longer superficial, but has already penetrated into the cord muscles or to the cricoarytenoid joint. As to the technic of the operation nothing new need be added. I would only recommend that, after the tumor has been removed by any of the aforesaid methods, the spot should be thoroughly cauterized with the galvanocautery. Local radium application after the operation, which has lately often been applied for prophylactic purposes, seems to have been beneficial.

(2) THYROTOMY.

This method is said to have been first used by Solis Cohen in 1868 for laryngeal carcinoma, but has come into vogue only later on the recommendations of Felix Semon, Otto Chiari, Hansberg, Ch. Jackson, etc., in the early forms of laryngeal cancer. Felix Semon, indeed, prefers this thyrotomy with the excision of the soft parts to the endolaryngeal operation recommended by Fränkel under similar indications, as not being dangerous and prognostically much better and much more favorable for the preservation of the voice. He was also of opinion that the estimation of the extent of the infltration could not always be properly ascertained by laryngoscopy, and the excision of the tumor by the endolaryngeal operation is not always sufficient. But I still maintain, that a smaller operation should, if possible, be resorted to, if it can be presumed that all diseased parts can properly be removed. Thus, I have had little opportunity to use thyrotomy, because I have employed the former method, where other authors resorted to thyrotomy. It is only when the cord carcinoma had, notwithstanding its early stage, already penetrated into the ventricular membrane or to the ligamentum thyreoarytenoideum or motility disturbances had already appeared that I performed this operation in 12 cases in all. Four of these were cancroids of the right or left vocal cord, three adenocarcinoma of the ventricle, two cancroid of the vocal cord and ligamentum thyreoarytenoideum, and one case each of medullary cancer of the left cord and adenocarcinoma of the left ligamentum thyreoarytenoideum. The results of our operations were not so good as those reported by Semon and Chiari. Five cases showed local recurrence, two of which were afterwards treated with total extirpation and the remaining three were lost sight of; two cases ended fatally, one from infectious pneumonia, the other on account of general debility after a long treatment for granulation proliferation on the wound. Only two cases proved entirely cured (one for over five years, adenocarcinoma of the left ventricle, another for over two years, cancroid of the right cord). The remaining three cases left our hospital in good state, but could not be kept under observation. The reason that my cases of thyrotomy gave inferior results is probably that they were much more unfavorable from the beginning, for the lighter cases were all treated endolaryngeally, where others would have applied thyrotomy. Under these circumstances I consider my results fair enough, considering that there already had been extensive infiltration, and yet two definitely and three relatively cured cases could be shown. The technic of the operation does not call for any remarks here, except that thyrotomy was always easily performed with cocain or novocain infiltration, sometimes combined with subcutaneous injection of pantopon scopolamin solution, and that the bleeding in operating the cordal carcinoma was never severe, so that I never was obliged to ligate the arteries or to apply Trendelenburg's or the sponge canula. I always performed tracheotomy shortly before the operation proper and the wound of this was always provided with the ordinary tracheal canula, which was removed after one week. The laryngeal cavity after the splitting of the thyroid cartilage was immediately painted with cocain adrenalin solution, and tamponed with iodoform gauze for a day after the operation. The further treatment was without tamponade.

(3) PARTIAL RESECTION AND (4) SEMILATERAL EXTIRPATION OF LARYNX.

Both methods were formerly frequently resorted to in the treatment of laryngeal cancer, seldom with good results. I have, therefore, always been of opinion that sharply defined semilateral or localized cancers, as such of the epiglottis, which had penetrated rather far, but which appeared as if easily removable by semilaryngeal or partial resection of the larynx, should, from the point of prognosis, at once be totally extirpated, because in both procedures the prophylactic precautions against pneumonia are insufficient, and the inclination to recurrence is very marked. For these reasons I have found few occasions to make practical use of any of these methods. But induced by Gluck's report, which he made in 1913 at the meeting of German naturalists and physicians in Vienna, that in 49 cases of hemilaryngectomy he had only three deaths, but 21 complete cures, I used this method in two well defined cases of semilateral cancer, but unfortunately lost both, one by pneumonia after one week, the other through recurrence after five months. Partial extirpation I performed three times, twice for carcinoma of the epiglottis by using subhyoid pharyngotomy. Of these one, the last was successful, in which I, after removing the epiglottis tumor, sewed the wound surfaces of the basis of the tongue and the aryepiglottic folds together to prevent pneumonia. After three months, by a small reoperation, communication between the pharynx and the larynx was again established and the outer subhyoidal opening closed. The other two cases, in which after extirpation of the epiglottis tumor I closed the outer wound with sutures, ended fatally through pneumonia. These experiences confirmed me in my opinion of the impracticability of hemilaryngectomy and partial extirpation of the larynx.

(5) TOTAL EXTIRPATION OF THE LARYNX.

Notwithstanding the good results of thyrotomy and laryngofissure obtained by such renowned specialists as Felix Semon, O. Chiari, Ch. Jackson and others, and of the endolaryngeal operation by Fränkel and myself, they can only be used in a limited number of cases, as cancer of the larynx usually comes to the specialist in already relatively advanced stages. Therefore, the principal operation will always remain the total extirpation of the larynx, as only in this way can all pathologic processes in the larynx be radically removed, and by further improvement of this method the inoperable cases may in future be more and more lessened in number. After Billroth first tried it in 1873, many famous surgeons followed him, but as you know, the results of these operations were at first not at all good, though, as Sendziak's statistics show, the fatal cases have become fewer in the course of time, and some complete cures were even established by this method (Obalinsky, Solis Cohen, etc.). This, on the one hand a pessimistic view came into vogue, which, supported by statistics, repeatedly asserted, that humanity would lose nothing by giving up this method, which was not to the credit of surgery; on the other hand, it is undoubtedly true that in recent times the total extirpation of the larynx carcinoma is quite frequently practiced by surgeons and by many laryngologists and that the results have become increasingly better. This progress has no doubt gone hand in hand with that of aseptic surgery in general, but for the progress in this special line we are chiefly indebted to Gluck of Berlin. In 1881, at the Surgical Congress in Berlin, Gluck published his experimental treatises, in which he showed that, in extirpating the larynx, or to express it more generally, in all operations, which so often cause death by pneumonia through foreign bodies entering the air passages, an absolute guaranty against this form of pneumonia could be established by prophylactic resection of the trachea and circular sewing up of the central stump of the trachea to the skin. In the years following he practiced this method with excellent results on many patients, and thus Gluck's method of total extirpation of the larynx has been brought to its present standard of excellence. The principal procedures of it are: (1) Complete exposure of the larynx before opening the air passage; (2) severing the larynx from the pharynx; (3) immediate closing of the pharynx defect while the larynx is drawn far out of the wound; (4) severing the larynx from the trachea; (5) circular sewing in of the lower stump of the trachea into the skin wound, and (6) closing of the whole wound by sutures.

Nineteen years ago I tried this method for the first time, but an unexpected difficulty made me modify Gluck's method to some extent, which, however, also gave satisfactory results. The patient was a merchant, 51 years of age, who suffered from internal carcinoma of the larynx. The left sided infiltration had already extended over the anterior commissure to about one-third of the right larynx. The symptoms were severe hoarseness and a moderate feeling of stenosis in the larynx. I intended to follow Gluck's procedure strictly, but during the chloroform narcosis symptoms of suffocation suddenly set in, which induced me to alter the operation somewhat. After the T-shaped epidermis cut in front of the larynx had been made I immediately amputated the latter below the ring cartilage by a cut slanting upwards from front to back, after severing the thyroid cartilage from its surroundings, applying a ligature at its isthmus in two places, cutting through and freeing the trachea where the resection was to take place with two raspatories from both sides and from the esophagus. Then followed Gluck's sewing of the lower tracheal end into the skin, in order to reestablish the breathing under narcosis with less danger and to completely prevent blood or mucus from entering the trachea. Then the larynx was freed from below upwards near to the upper rim of the ring cartilage from the esophagus to the frontal hypopharyngeal wall, partly by blunt, partly by sharp dissection, and the cornu superius of the cartilage was cut off on both sides with scissors. Until then the pharynx had not yet been opened. After the whole wound, especially the part of the hypopharynx and the esophagus and all exposed muscles had been covered with thick gauze strips the pharynx was opened subhyoidally and immediately tamponed with gauze to prevent flow of secretions and infection of the wound surface. Then followed the severing of the larynx from the pharynx and the sewing up between the root of the tongue and the upper stump of the front wall of the esophagus or hypopharynx. Finally, after making counter openings on both sides of the tracheal opening, the outer subcutaneous wound was thoroughly tamponed with iodoform gauze and the epidermis ends were sewn together. For one week only liquid food was given by means of a permanent esophagus catheter. The convalescence was excellent, all sutures healed per primam intentionem. After ten days food could be taken without the catheter and after two months the patient could be discharged cured. He was subsequently demonstrated at the monthly meeting of the Japanese Oto-rhino-laryngological Society together with the removed parts. The man is still living and, considering his age, 70 years, quite well and active. It should be added that he can speak in whispers and make himself understood, in the beginning with the aid of Gluck's rubber tube, but later without it. Encouraged by this success I have up to December of last year (three cases in this year cannot be counted in yet) performed the same operation in 106 cases of various forms of laryngeal carcinoma, both internal and external forms, with and without accompanying glandular metastasis on the neck, with the following results:

Thirty-six cases showed no recurrence after three years, and fifty-five cases relatively cured, including all successful cases within the last three years and those who were discharged cured but have been lost sight of. Ten cases recurred, two of them locally near the epiglottis, one at the thyroid, which latter was completely cured after reoperation; in seven glandular returns appeared, some of which could be relatively cured by reoperation.

Six cases ended fatally within one to four weeks after the operation; one on the tenth day through erysipelas, another on the seventh day through uremic anuria; one case ended on the twenty-fifth day on account of general debility and three with infectious pneumonia. The infection was, however, not caused by the influx of blood into the trachea, but later (two cases within ten days, one after two weeks) on account of local suppuration of the wound.

Compared with Gluck's, my results do not appear so good, but I must mention that I have ventured to operate on cases too which, according to Cerny and others, were until now considered inoperable, on aged persons of 70 years and more, on patients who already showed glandular swellings on one or both sides of the neck, in the hope of reducing the number of inoperable cases as much as possible. Taking all this into consideration, I think I can be satisfied with my results. Moreover, the causes of death in these six cases were not the effects of the operation itself, but mostly some accidental neglect, which with greater watchfulness on the part of assistants and nurses may be avoided. I therefore feel myself justified in recommending the above method to you, adding the following critical remarks:

The modus operandi adopted by me is really not new. From remarks made by Gluck I know that a French specialist, Lubec, long before Gluck, performed a very similar operation, by resecting the trachea in the first stage and sewing its lower end into the skin, letting the principal operation follow a fortnight later. Here in America James Bell is said to have reported in 1909 eight successful cases, which he had operated on in a manner very similar to mine, saying: "Without previous tracheotomy the larynx is removed from below upwards, after sewing the trachea to the skin." It is really not my intention to raise here the question of priority. I only wish to state that as early as 1903 I used this method on a patient, on whom I intended to perform Gluck's total extirpation, when some difficulty accidentally arose on account of the patient, and that since then I have practiced this method for nineteen years in more than a hundred cases of laryngeal carcinoma, finding that it is easier to perform and less risky, and that it provides the same protection against infectious pneumonia as Gluck's method. If it should be objected by Gluck that the procedure of laryngectomy—that is, the total extirpation of the larynx from below upwards—increases the risk of infection on account of the early opening of the trachea, I would say on the contrary that the earlier opening of the pharynx cavity, which is the essential part of his procedure, is a source of danger from wound infection. For even if the mouth and pharynx cavity are at once tamponed with long strips of gauze and the wound surface likewise packed with gauze, very often quantities of mouth or pharynx secretions exude from the cut with reflex retching or swallowing movements. This not only greatly hinders smooth working but can frequently give opportunity for infection. In this respect I think my way to be even better, because the opening of the pharynx cavity comes last, after the trachea and the larynx have been completely separated from their surroundings and all wound surfaces have been thoroughly tamponed. For a very short time only the pharynx cut remains open, for immediately after tamponing the mouth and pharynx cavity with gauze or sponges sutures are made between the base of the tongue and the upper rim of the anterior hypopharynx. Moreover, the early resection of the trachea and the sewing of its lower stump to the epidermis I consider to be not at all so dangerous as Gluck believed, because the breathing opening is thus far removed from the chief field of operation. The following points I carefully heeded during the operation:

(1) The operation must be made under the strictest aseptic and general surgical rules.

(2) It may often be performed under local anesthesia, as nearly all Austrian, French, Italian and other specialists have expressly recommended. I myself have used successfully in six cases novocain infiltration combined with previous injection of pantopon scopolamin. But considering that with such a long and complicated operation a general chloroform narcosis is much more convenient, because the patient very often still feels pain after simple anesthesia, and that the injection of pantopon scopolamin is not always harmless, as P. Heyman

has asserted, I have mostly used general chloroform narcosis and have never had any bad effects.

Gluck formerly recommended the formation of a large winged lap in front of the larynx for initial skin incisions, and later a median longitudinal cut from the level of the hyoid bone to the jugulum and two horizontal cuts at the level of the hyoid bone at the lower edge of the ring cartilage from the forepart of one sternocleidomastoideus to the other, as being best for opening up the field of operation in all its parts and to make a reliable drainage of the wound cavity possible after the operation. I prefer the T-shaped cut between the hyoid bone and the fossa jugularis, by which I could sufficiently fulfill Gluck's two conditions, and which in some respects seems to me even of greater advantage, inasmuch as the tracheal stump sewed in need not necessarily be freed from its surroundings but can remain in firm connection with it.

(4) In resecting the larynx any tearing or injury to the surrounding muscles, fasciæ, vesicular sheaths, etc., must be avoided as much as possible.

(5) The thyroid artery, if still quite healthy, is better left in by ligating it in the middle of the isthmus in two places and cutting through between. If, however, the carcinoma has already penetrated into the neighborhood of the gland and there is any suspicion of its being affected, it may likewise be removed. I have not been able to confirm that the removal of this gland is in any way harmful to the organism.

(6) As Gluck has already fully explained, I too advise the removal of the regional lymphatic glands and ducts Only in cases of well localized intrinsic carcinoma without glandular swellings I leave these parts alone. But in cases where the lymphatic glands already show swelling, or in extrinsic cancer I carefully try to remove all existing glands, even the smallest, together with all lymphatic vessels lying between the diseased part and the muscles and fasciæ. Cases where the metastatic glands are already of considerable size and adhere to the large vessels of the neck must be declared inoperable.

(7) I will not enter into details about the after-treatment, but make only some few remarks:

(a) The gauze strips left in the wound cavity are removed after one week if nothing untoward has occurred until then, and replaced by small drainage tubes.

(b) For feeding I frequently introduce the small permanent catheter through the nose, or in some cases two or three times a day the usual esophagus catheter orally into the stomach. But I prefer the former, as there is danger of piercing the pharyngeal sutures with the latter.

(c) If the healing process progresses well, the permanent catheter is removed after one week and the patient can be fed in the ordinary way. If the pharyngeal sutures have opened, however, the catheter must be left in longer.

(d) If the strength and the state of the patient permit it, it is advisable to make him get up as soon as possible and even let him walk about the room.

GENERAL REMARKS AND CONCLUSIONS.

From all the facts presented here we can clearly see that we are in the period of achievement as regards the treatment of laryngeal cancer, and that Virchow's words have been realized: "If cancer is, in its beginning and for some time longer, a local affection, it must be possible to cure it locally at this stage."

And there is no doubt that the earlier the beginning carcinoma of the larynx is diagnosed and treated by a suitable operation, the more likely the success of the operation, as Fränkel and others have repeatedly said. We also know that the prognosis of laryngeal cancer has, with the progress of laryngeal diagnosis and with the development of suitable operating methods, going hand in hand with the general progress of surgery, become greatly improved, as recent statistics show. But if we compare the number of cases which have been treated successfully, either by operation or otherwise, with those not operable or operated on unsuccessfully, it becomes clear that we must not rest satisfied with what we have achieved. In our university clinics, where roughly 8,000 patients annually are taken in, and in my private hospital, I have seen within these last 20 years over 400 cases of laryngeal cancer, of which the majority for various reasons could not be operated on, partly on account of extensive glandular metastases, partly because of severe cachexia or general debility, or on account of coexistence of other diseases, affections of the kidneys, heart, lungs, etc., and often also because of the refusal of an operation by the patient or his relatives. Our aim must be to improve the prognosis. To this end I wish to express our desiderata in the following concluding sentences:

(1) We must instruct the general public and practicing physicians by writing and by speech, that laryngeal cancer may begin without a known cause and without severe symptoms, sometimes with simple hoarseness or with slight stinging pains in the throat, symptoms which may probably be diagnosed by the ordinary physician as nothing more than laryngitis simplex, and that just this beginning form, prognostically and therapeutically, is most important, for it is certain that this form can with great probability be cured by a comparatively simple and harmless endolaryngeal operation or by thyrotomy.

(2) Practicing physicians, family and companies' doctors often have opportunities of observing these beginning forms and should become at least as familiar with laryngoscopic as with stethoscopic examinations. To attain this end I would propose to improve the methods of teaching laryngoscopy and to raise the position of laryngology in the realm of medical science.

(3) We specialists should in all suspicious cases endeavor to make an exact clinical diagnosis as early as possible, combined with microscopic examination of a test piece which has been immediately cut out after the laryngoscopic examination. If exact diagnosis shows the case to be one of cancer, a suitable method of operation must be selected according to the exactly ascertained nature of the case.

(4) We must not mechanically stick to only one method of operation, but must be able to individualize and prefer the smaller operation to the larger; but in case the indications should point to the necessity of the larger operation, we must not hesitate in employing the same.

(5) For selecting a suitable method I would propose my modus, which has proved practicable by long experience: Sharply defined polypoid forms on the free edge of the vocal cord, which have not yet penetrated into the ventricular membrane, or the movable forms of the pedunculated carcinoma are best operated on by the endolaryngeal excision of the

tumor, of course by a well practiced hand, with indirect method or with the suspension laryngoscope. In case these localized forms of the laryngeal cancer have already penetrated deeper into the ventricle, or in cases of primary ventricular forms (mostly adenocarcinoma), or if the surgeon is in doubt as to his dexterity for an endolaryngeal operation, I should recommend thyrotomy with subperichonderal excision of the tumor afterwards.

In cases where the carcinoma has not only affected the vocal cords but also the arytenoid cartilage or the cricoarytenoid articulation, or the posterior wall of the larynx, or has progressed over the anterior commissure to the other side, total extirpation must immediately be resorted to, if the constitution of the patient permits.

Partial resection of the larynx and hemilaryngectomy are, according to my experience, not very practicable; they may even prove dangerous, as it is difficult to separate the wound cavity from the air passage by a tissue wall. I would recommend this method only for sharply defined epiglottis carcinoma, as we can insure security against infectious pneumonia by a temporary closure of the introitus larynx by the suturing of the base of the tongue to the upper rim of the posterior wall of the larynx. Hemilaryngectomy would be better replaced by total extirpation.

This brings my short exposition to a close. Gentlemen, I thank you for the patience with which you have listened to my inadequate remarks on the subject.

XXXIX

THE STUDY OF THE TONAL RANGES IN LESIONS OF THE MIDDLE EAR.

By L. W. Dean, M. D., and C. C. Bunch, Ph. D.,

Iowa City, Iowa.

The research work which we are reporting represents a large amount of careful, conscientious work, especially on the part of Dr. Bunch, who either made or supervised all the functional tests of hearing which are indicated. This report is confined to the examination of the auditory perception of a large series of cases of so-called middle ear trouble. In all cases exhaustive clinical examination was made. These examinations were grouped and studied, and the results are here summarized and presented for your consideration.

Plate 1 is the diagram of the clinical record made for each case examined. In only a few cases where it seemed desirable to have specific information regarding the vestibular functions were the turning tests made.

The hearing for whispered and spoken voice was first determined. Next the pitch range audiometer was used to determine the audition for tones between 30 and 7070 d. v.

The records with the audiometer are easily indicated by means of a graph shown in the plate. The pitch of the tones is indicated at the bottom and the loudness of the tone by the height of the curve on the graph, no tones at all being normally heard at zero. The curve shown on the graph is the field of hearing for a normal ear.

In the curves of cases studied that we are going to show, the curve was secured by testing the audition of the ear examined for every tone from 30 d. v. to 7070 d. v. The curve thus secured compared with the normal curve, which is stamped on every chart, gives an approximate idea of the relative hearing power for these tones.

If, for instance, the curve at 1,000 d. v. is only half as high as normal, this does not mean that hearing for that note is one-half normal but only that hearing for the note is markedly decreased. We are not justified in assuming that the curves represent the exact audition for the note that is being tested. The fractions which express quantitatively the audition for various notes, when this is determined with the Bezold forks, are likewise very inexact. As the intensity of sound varies with the square of the distance, with the square of the amplitude of the vibration, and is influenced by the rapidity of vibration, it is an extremely difficult matter to determine of vibration of sound, it is an extremely difficult matter to determine in an yway the exact amount of audition for any given note.

If the curve of the ear being examined should show a tone gap in the tonal range, this does not necessarily mean that there is an absolute tone gap but it does indicate that the ear is unable to perceive the loudest tones capable of being produced by the audiometer. These gaps found by the audiometer have been verified in the psychologic laboratories by tests with organ pipes and other instruments producing pure loud tones. It is easy to conceive that if a note could be produced of greater intensity than that which the audiometer can give, it might be heard when the audiometer would produce no sensation of sound.

In the following charts the normal curve is indicated by a continuous line; curves for the right ear by dashes; and for the left ear by dots.

For the benefit of those who are not interested in the audiometer readings we have made in each case a quantitative determination of the audition for the 50 d. v. fork, the c, c^1, c^2, c^3, c^4, and c^5 forks.

The Rinne, the Weber, and the bone conduction tests were made with the 256 fork. In some cases the 128 and 512 d. v. fork was also used. These forks were weighted to minimize the production of confusing overtones.

At the bottom of the plate next to the audiometer curve is a measurement of the upper tonal limit, as determined with the Galton whistle, the Koenig cylinders and the monochord. The upper limit of the monochord is determined both by A. C. and by B. C. With the Galton whistle which we use the normal upper limit is 21,000 d. v. With the Koenig cylinders the upper limit is 43,960 single vibrations and with the monochord by A. C. 19,000 d. v., by B. C. 20,000 d. v.

In each case studied we have been interested in determining if in addition to the middle ear lesion there was also present a lesion of nerve or end organ.

Suggestive of a lesion of the acoustic nerve or its end organ is the following:

1. The audition for the higher notes is poorer than that for the lower or middle part of the tonal range. If the lower notes of the tonal range up to about 3,000 d. v. are heard normally and there is a diminution in the audition for the notes in the upper part of the range, as is shown by Plate 2, this diminution of audition for the higher notes is suggestive of a lesion of the nerve or end organ. If, however, as is shown in Plate 3, there is a uniform diminution in audition for tones throughout the range, it does not indicate a lesion of nerve or end organ. The curves shown in Plate 3 are the curves secured from: first, a case of impacted wax in the external canal; second, a similar case; third, three cases of individuals with normal hearing where the external canal has been packed with boric acid powder, and lastly, a case of cotton in the ear which was soaked with cerumen and was in contact with the drumhead. In each of these cases you note the diminution in audition for all tones throughout the range. As we view it, there would have to be a distinctly greater diminution for the notes in the upper part of the range as compared with the tone best heard in the lower or middle part in order to indicate positively a lesion of the nerve or end organ. In some of these cases the c^5 fork was not heard at all.

Plate 4 shows the curves of these cases after the removal of the impacted wax, cotton or boric acid powder. Note that the curves are practically normal.

2. A lowering of the upper tonal limit, especially by B. C. The better the hearing for voice and whisper, the more significant of a lesion of nerve or end organ is the decrease in the upper limit by A. C.

Plate 5 indicates the upper tonal limit in three of the cases of obstruction in the external canal just described by A. C. and B. C. determined with the monochord. Note the upper limit by A. C. is lowered 2,000 d. v. from obstruction in the external canal. There is, however, no cutting down of the upper limit by B. C., and we feel that a lowering of the upper limit by bone conduction is a more definite indication of a lesion of nerve or end organ than the lowering of the upper limit by A. C.

Plate 6. The comparison of the upper limit of audition for high notes as determined by A. C. and B. C. is of interest.

The upper chart shows the A. C. and B. C. as determined with the monochord for twenty-two acute suppurative ears. The vertical figures represent the number of cases; the horizontal the upper limit in double vibrations per second. The curves are similar, but by air the lowest limit was 12,000 d. v., while by B. C. it was 14,000.

The results of examination of twenty-eight cases of chronic otorrhea are charted in the center. Again the larger number of cases make similar curves, but here, while the lowest limit by B. C. is 13,000 d. v., by A. C. it is 6,000—a great difference.

Below are graphs secured for charting the results of forty cases of hyperplastic otitis media. Here the similarity of the curves is lost.

The diagnostic significance of the difference between the upper limit as determined by bone and air conduction with the monochord is here illustrated. In cases of acute trouble 22 per cent gave a difference of 23,000 d. v. or more between the upper limits determined by air conduction and by bone conduction. In the chronic otorrhea cases this percentage was 39, while cases of hyperplastic otitis media gave this difference in 67 per cent.

The determination of the upper limit by B. C. is a far more accurate indication of the condition of the nerve and end organ than its determination by A. C.

3. Positive Rinne.

4. The sensitivity for one note or a group of notes is much less acute than the others when determined with the tuning fork or the audiometer.

To determine whether there is a significant difference between the hearing power for high tones and low tones, we have used the audiometer and the Bezold forks. For example, if the hearing for the c^2 fork is indicated by the fraction 20/24 while that for the c^5 fork is indicated by the fraction 5/60, there is a greater decrease for the c^5 than for the c^2. This would be shown on the audiometer record by a curve which closely follows the normal curve for a frequency of 512 d. v., but which falls much below the normal curve at 4096 d. v.

Plate 7 is a plate of neurolabyrinthitis showing these distinct depressions in the audiometer curve confirmed by the findings with the tuning forks.

5. Decreased bone conduction as determined by the c^1 and c^2 fork.

This is a very important matter. We feel that a bone conduction decreased two or three seconds is suggestive of a lesion of nerve or end organ when the examination had been properly made. We test bone conduction by placing the tuning fork over the bone just adjacent to the external opening of the bony canal posterior to the upper attachment of the external ear. We keep in mind the influence of mastoid involvement decreasing the bone conduction.

Bezold (Textbook of Otology, page 67) thinks that to be significant the perception by bone conduction for the fork placed on the vortex should be very much shortened.

Wittmaack (Zeit. f. Ohr., vol. 60, page 136) is of the opinion that a shortening of the bone conduction is worthy of diagnosis when it amounts to at least 30 per cent of the average value which we get or is received through the examination of a large number of normally hearing individuals with the chosen tuning fork.

As the percentage of decrease will depend altogether upon the energy with which the fork is excited, this statement is of little value.

Bezold feels that an objection to this procedure is that extraneous sounds and the ability of the individual to make an accurate examination and of the patient to cooperate have a decided bearing upon the result. Such criticism is valid for all functional tests of hearing and is as true for determining hearing power by A. C. as well as B. C.

Our examinations are conducted in a room set apart especially for this purpose and, while it cannot be said to be entirely free from external noises, it is probably much more nearly ideal for this purpose than those ordinarily used.

Emerson (ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Dec. 21, page 999) is of the opinion when B. C. is lowered for the 256 or 512 d. v., that fork nerve deafness has been present for a long time.

Werhofsky (Prufung. d. Herdauer in Verlaufe der Tonskala bei Erk. d. Mit. u. Innern O. Bezold U. d. Funk, Pruf. d. Mench. Gehor., 1897-1909) found decreased B. C. in all lesions of the nerve or end organ that he studied. He reports B. C. decreased two seconds in one case of nerve deafness and three seconds in another.

He says that B. C. is less with higher forks than with lower and cites a number of observations to confirm this.

In describing his technic, he does not state the length of time his forks vibrate with a given stimulus. Our higher forks vibrate longer than the lower, and consequently the decrease in B. C. with the higher forks would be greater.

Siebenmann in an article translated by Dench (Archives of Otology, vol. 22, p. 5) says that in apparently normal ears there may be functional defects in the test for the upper tone limit and in the perception of sound by B. C. He also shows that B. C. is sometimes decreased by aspiration of the middle ear.

Page (Proceedings N. Y. Acad. of Med., Oct. 13, 1921) says that the early cases of otitis media have a negative pressure in the tympanum.

Kerrison (Text Book of Otology, pg. 89) suggests that the hearing by bone should be decidedly decreased to be significant.

Schwabach (Uber Harprufung, etc., Archiv. f. Ohren., vol. 31, page 6) says that B. C. is longer than with normal persons in lesions of the transmitting apparatus, while it is the same as normal persons or less in lesions of the perceiving apparatus of the ear.

In our study of acute middle ear infections we have found it very unusual to find an infected ear without evidence of a lesion of its nerve or end organ.

Siebenmann, in an article translated by Dench (Archives of Otology, vol. 22, page 12) reports that in making functional examinations of pure cases of catarrh of the eustachian tubes he found the upper tone limit usually lowered and not restored by inflation. He assumed that the lowering of the upper limit was due to a passive hyperemia of that portion of the scala vestibuli lying next the oval window.

Plate 8 shows curves of a case of acute tubal catarrh where we found no definite evidence of lesion of nerve or end organ. The decrease in hearing for the voice and whisper was apparently due to the middle ear condition and disappeared wholly on inflation. B. C. in each ear was normal. Upper limit by monochord, R., A. C. 16,000, B. C. 19,000; L., A. C. 17,000, B. C. 19,000. As with the blocking of the external canal the upper limit was lowered by air and not by B. C. Unfortunately, after inflation the upper limit was not tested by A. C.

The curve before inflation shows rather a uniform decrease in audition for all notes except the very lowest, which were not heard at all. The audition for the notes became almost normal on inflation.

Twenty-two acute infected ears were studied. Of the twenty-two, fourteen had positive Rinne and eight had negative Rinne; fifteen had decreased bone conduction for the c^2 fork and fourteen had the upper tone limit decreased 2,000 or more by A. C. as determined by the monochord. Thirteen of the fourteen had the upper limit by B. C. decreased 2,000 or more. Twelve cases had decreased bone conduction as well as lowered upper limit. Two cases had decreased B. C. with upper limit normal and two cases had upper limit by A. C. lowered but B. C. normal.

Plate 9 shows the curves of ten acute infected ears. The dots, dashes and circles in the curves are added so that they may be more easily followed through the entire field. You will note that curves Nos. 2 and 3 show complete gaps. Partial gaps are shown in all the curves except No. 4. Ninety per cent of the curves of acute infected ears are suggestive in this manner of lesions of the nerve or end organ.

A careful analysis of the clinical charts of the twenty-two ears studied shows that in every acute infected ear except one there was either decreased B. C., upper limit lowered by B. C. 2,000 or more, or a defect in the audiometer curve suggestive of lesion of nerve or end organ.

These evidences of lesion of nerve or end organ appear very early in the course of the disease.

Plate 10. H. B. Examination was made within twelve hours after first evidence of infection. With good hearing for voice, spoken, 45 feet, and whisper, 15 feet, in each ear; frank earache in the left, slight earache in the right; drumheads reddened, no bulging; Rinne positive in each ear, c^5 fork in left 30/60; with other forks normal. Two partial tone gaps in left ear with audiometer curve. B. C. 2 sec. each; upper limit lowered 2,000 by B. C. with monochord. No leucocytosis at this time. Patient discharged ten days later, B. C. normal; c^5 heard normally, upper limit normal by A. C. and B. C.

In cases where the infection was unilateral the evidences of a lesion of nerve or end organ were not confined to the infected ear.

Plate 11 shows the findings of the good ear in six cases of unilateral acute infection of the middle ear. Only one case, the first, had no suggestion lesion of end organ or nerve in the good ear. In the second, third, fourth and fifth cases the upper limit is lowered; in the fourth, fifth and sixth B. C. is decreased. The audiometer curves of the first case, and of cases three and four, were somewhat suggestive of lesion of nerve or end organ so that in at least five out of six of the unaffected ears, and probably all six, there is evidence of involvement of the nerve or end organ.

Plate 12 B. D. This case is interesting because a few days before infection of the left ear the ears were tested and were apparently normal. Note that in the right ear where there was no redness of drumhead or other evidence of middle ear infection, the decreased hearing for voice and whisper, the decreased B. C., the decreased upper limit by B. C., and the slight diminution for the c^1, c^2, c^3, c^4 and c^5 forks, for the left ear, the decreased B. C., the tuning fork tests and the audiometer curve are all suggestive of lesion of nerve or end organ.

Treatment and eradication of the middle ear condition may have no influence upon the lesion of nerve or end organ.

Plate 13 E. B. shows the findings at the height of an acute tubal catarrh. The tubal orifices were edematous, covered with dilated blood vessels; the drumheads were reddened, not bulging. B. C. decreased each 2 secs. Upper limit each B. C. 17,000 by monochord.

Plate 14 shows the findings after the eradication of the middle ear lesion. B. C. —3. Upper limit lowered by B. C. Audiometer curve indicative of lesion of nerve or end organ. The findings remained the same as long as the patient remained under observation.

Plate 15 shows the findings A. A. S., taken one month after an infection of the left ear was apparently well. Curve and tuning fork findings for left ear indicate a lesion of importance involving nerve and end organ. Whisper and voice are poorly heard.

Plate 16 B. D. shows findings in both ears in a case of acute infection of the left ear one month after discharged as cured. The curve left, tuning fork tests left, and decreased B. C. each indicates a lesion of nerve or end organ left and probably also right. At no time during the disease was there evidence of acute middle ear infection right. Nevertheless, the hearing right which was W15, V45, before the infection of the left ear is now W13, S45, while the B. C. is —5. Before the infection of the left ear it was normal.

After acute infections of the middle ear with return to normal audition for voice and whisper there may be definite indications of lesions of nerve or end organ.

Twelve cases of bilateral chronic otorrhea and fifteen cases of unilateral chronic otorrhea, or thirty-nine ears, suffering with chronic otorrhea, were studied. Rinne was negative in thirty-five ears, positive in four. There was decreased bone conduction in eleven ears, increased in twenty-six, normal in two. Decreased B. C. was not so common as in the acute infections. The upper tone limit was decreased 2,000 or more by bone conduction in thirty ears. In thirty-seven ears the audiometer curve was suggestive of a lesion of nerve or end organ and in two ears not. Marked depressions and tone gaps in the audiometer curves were much more frequent than in the acute ears. The curves were much more characteristic of lesions of the nerve or end organ. If we include the results with the tuning fork tests, in only one ear out of the thirty-nine was there no evidence of a lesion of the nerve or end organ.

Plate 17 R. B. is a typical chart showing the findings in a case of chronic otorrhea. Note that with the tuning forks the

hearing range is from c^1 to c^4 inclusive. The audiometer curve shows the decreased audition for both low and high notes in the range of the audiometer with depressions characteristic lesions of the nerve or end organ. The upper limit in each ear is decreased to 15,000 d. v. or B. C.

From a study of the cases of chronic otorrhea one gets the impression that the extent of the lesion of nerve or end organ corresponded roughly with that of the middle ear lesion, while in the acute cases there was the greatest variation between the extent of the lesions of the inner ear and those of the middle ear. It was not unusual with a very mild acute middle ear infection to have a very well marked lesion of the nerve or end organ. A study of the fifteen unaffected ears in the fifteen cases of unilateral chronic otorrhea shows that Rinne was negative in unaffected ears no times, positive in fifteen. B. C. was decreased in the unaffected ears three times; it was normal ten times and increased twice; the upper limit was decreased by B. C. 2,000 or more five times and was normal ten times. The audiometer curve of the unaffected ears was indicative of a lesion of nerve or end organ in only one case. The unaffected ears in this series of unilateral cases were very good ears with one exception. The hearing in almost every ear was W 15 feet. While these findings suggest that there are a fair number of lesions of the nerve or end organ in the unaffected ears in chronic otorrhea, we do not find the large percentage of involvement of the nerve or end organ as in the unaffected ears in the acute infections. This suggests to us that the involvement of the nerve or end organ in the unaffected ears in acute infections is frequently not a permanent affair; otherwise, we would find the same number of lesions in unaffected ears in chronic otorrhea. The lesions of the nerve or end organ in ears suffering from chronic otorrhea were much more marked than in the acute infections. There is a direct interdependence between the chronic otorrhea and the lesions of the nerve or end organ. The chronic otorrhea has no influence of any moment upon the nerve or end organ of the opposite side. In our acute cases we get the impression that the acute middle ear trouble and the lesion of the nerve and end organ are not interdependent. They may both be due to the same cause but not necessarily so.

Plate 18 is a composite graph of the results obtained from tuning fork examinations. It is difficult to express in a uniform manner the results obtained in tuning fork examinations when different denominators are used to represent normal hearing for different tuning forks. For example, in measuring the hearing power for the tuning forks we represent the hearing for a normal ear in terms of distance as the denominator of a fraction. The hearing power for that fork by a pathologic ear is recorded in the same terms in the numerator. For the c^5 fork, since a very slight impact produces a sound heard at a considerable distance, we use the denominator 60. The 256 fork is heard at a less distance and we use 24 for the denominator. For similar reasons we use the denominator 4 with the 50 d. v. fork.

In order to make an analysis of our results we have arbitrarily divided our results into six classes. Those who do not hear the fork at all are put in Class 1; those who hear the fork when it is very loud, as indicated by fractions equivalent to one-fourth or less, we have put in Class 2; those who hear it by values indicated by the fractions between one-fourth and one-half are put in Class 3; those who hear it by fractions from one-half to three-fourths are put in Class 4; those who hear it from three-fourths to less than normal are put in Class 5; and those who hear it normally are put in Class 6. The curve shows that twenty-one cases did not hear the 50 d. v. fork at all; three did not hear the c^5 fork; fourteen heard the 50 d. v. fork with a fraction equivalent to one-fourth or less; while twenty-seven heard the c^5 fork with results indicated by a fraction of one-fourth or less. You will notice that the 512 d. v., 1024 d. v. and 2048 d. v. forks run almost parallel throughout the entire curve, while the greatest variations in chronic otorrhea cases are shown with the 50 d. v. fork and the 4096 d. v. fork. There were no cases where the hearing was perfect for the low fork and two cases where the hearing was normal for the 4096 d. v. fork.

Plate 19 shows the results of the audiometer examinations in ten cases of chronic otorrhea. It is inserted to show the frequency with which such phenomena as tone islands, gaps and other peculiarities exist. These peculiarities are indicative, in our judgment, of lesions of the nerve or end organ.

For example, curve No. 8 shows simply a region of residual hearing for tones between 500 d. v. and 1500 d. v. No. 7, while rising much higher on the chart, shows two islands with the interlying gaps at 2500 d. v. and at about 3200 d. v. Curve No. 9, even though rising to the normal at 1000 d. v., shows that the low tones are lost, that there is a decrease for notes above 1200 d. v., and a return to almost normal at 5500 d. v.

Sixty-six per cent of the records of cases with chronic otorrhea show either complete or marked partial tone gaps. These do not include such cases as are illustrated by curves Nos. 1, 2 and 8, which are equally significant of an inner ear lesion.

In only one case, L. P., age 13, did a tone gap disappear with treatment.

Hyperplastic Otitis Media.—Thirty cases of bilateral H. O. M. and four cases of unilateral H. O. M., or sixty-four ears, were studied. Rinne was negative in thirty-three ears, positive in thirty ears, questionable in one ear. There was decreased bone conduction in forty-four ears, increased bone conduction in sixteen ears, normal in four ears. The upper limit was decreased by B. C. 2,000 or more d. v. in forty-nine ears.

Twenty-one ears had complete tone gaps in the audiometer findings. In eight more there were marked partial gaps or 45 per cent showing these puecularities. In addition to this there were 34 cases where there were marked depressions equally indicative of inner ear involvement.

Plate 20 shows the curves of ten typical cases of H. O. M. Complete tone gaps are illustrated in curve No. 10, which has a gap at 2400 d. v. and another at 2000. Curve No. 9 shows a dip or partial gap at 1600 d. v. No. 6, while not showing either a partial or complete gap, shows that the hearing was normal up to 1500 d. v. and decreased more and more for tones higher in the scale. A curve of this type certainly indicates a lesion of the nerve or end organ. Curve No. 8 shows only a patch of residual hearing between 100 d. v. and 1500 d. v. Each curve shows marked diminution in the audition for the c^5 fork. In the next plate you will note that in only eight of the sixty-four ears was the c^5 fork heard normally.

Plate 21 shows a composite of the tuning fork cases in hyperplastic otitis media. Here we have made the same arbitrary divisions as we did with Plate No. 18. Here again we see that the larger variation is shown with the 4096 d. v. and the 50 d. v. forks. The 1024 d. v. and the 2048 d. v. forks run almost parallel through the entire range. While there were no cases where the 4096 d. v. fork was not heard there were 51 cases who could hear this fork only when it was struck very loud as, for instance, when the fork was forcibly struck against the heel of the shoe, twenty cases did not hear the 50 d. v. fork.

Note that in eight cases the 50 d. v. fork was heard apparently normal. In these eight cases the lesion of the nerve or end organ overshadowed the middle ear lesion.

A study of the clinical charts of these cases shows evidence of the involvement of the nerve or end organ in every ear affected save one.

Plate 22 F. K. shows the curves of a bilateral case of H. O. M. Note the negative Rinne, slightly decreased B. C., power of hearing low and high notes markedly interfered with in each ear, tone gaps in the audiometer curve, and upper limit markedly decreased both by A. C. and B. C.

Plate 23 C. L. shows in the right ear bone conduction normal, and negative Rinne, audiometer curve practically normal, upper limit normal. In the left ear B. C. is decreased five seconds. The right ear of this case was the only one of the sixty-four ears studied that did not show some evidence of involvement of nerve or end organ. Hearing was poorer in the right ear.

There was found no definite relation between the extent of the inner ear lesion as compared with the middle ear lesion.

Plate 24 shows the findings in H. S., aged 62, and in C. P., aged 62. The objective findings of the middle ear were similar. In one case the bone conduction is —5 seconds and in the second case it is —12 seconds. In the first case the audiometer curve shows a tone gap in the left ear and in the second case there is none. In the audiometer curve the upper notes are heard much poorer in the first than in the second. In the first case the upper limit is so low that it cannot be detected using the Galton whistles or the monochord, either by A. C. or B. C., and with the Koenig cylinders it is only approximately half normal, 21,000 s. v. instead of 41,000, while in the second case the upper limit is decreased, it is 16,000 by B. C. with the monochord. The lesion of the nerve or end organ is much more marked in the first than the second case. In the second case, the one with the least involvement of the nerve, had a positive Wassermann, the first one not.

Plate 25 (Findings of Miss D. and H. B.).

The age of the first is twenty-five years and the age of the second is twenty years. The hearing in the left ear is approximately the same for whisper. In the first case B. C. left is increased, the tuning fork test shows fair hearing for the c^4 and c^5 fork, the upper limit by B. C. is normal, the audiometer curves, while there is a gap in the right ear and depressions in the left, shows fair hearing for some of the notes in the higher part of the range. In the second case, however, B. C. for c^1 fork is —5, c^2 fork —6, hearing for the c^4 and c^5 fork is markedly decreased, the audiometer curves show a cutting down of the higher notes to 4,000, the upper limit by B. C. is 15,000. The evidence points to a much more marked lesion of the nerve or end organ in the second case than in the first.

In one case of H. O. M., while under observation acute suppurative otitis developed. With the diminution in hearing the B. C., which was plus 4 in the left ear, became decreased. With the disappearance of the acute otitis it became plus again.

Our cases of H. O. M. frequently tell of improvement in hearing that is satisfactory to them following one kind of treatment or another. Our tests have failed to show any actual improvement in any case unless the lesion of the nerve or end organ greatly overshadowed the middle ear lesion, and the improvement resulted from the treatment of the case for syphilis, or removal of tonsils, or what not.

Plate 26. H. J. is a case of H. O. M. where there was also a plus 4 Wassermann. Note the positive Rinne with the c^2 fork and the greatly decreased B. C. with the same fork and a negative Rinne with the c^1 fork, which is decreased only six seconds. This patient's deafness has rapidly appeared in the last six months. It has progressed too rapidly for H. O. M. The syphilitic lesion of the nerve or end organ is more responsible for the deafness.

Plate 27. J. C. is a case of H. O. M. with negative Wassermann and no evidence of syphilis, where the lesion of nerve or end organ greatly overshadowed the middle ear lesion. Note the Rinne plus, B. C. decreased 6 seconds and 4 seconds, the tuning forks suggestive of lesion of nerve or end organ. The upper limits by B. C. 12,000 d. v. and 13,000 d. v. The curve is also characteristic of lesion of nerve or end organ.

A study of the unilateral cases of H. O. M. gives some very interesting information. Four cases were studied. In the unaffected ear, negative Rinne in no cases. Decreased B. C. in two cases, upper limit decreased 2000 or more by B. C. in three cases. In all four of the unaffected ears there was some evidence of lesion of nerve or end organ if we include the findings with tuning forks and audiometer.

CONCLUSIONS.

1. The determination of the upper limit by B. C. is a far more accurate indication of the condition of the acoustic nerve and its end organ than its determination by A. C.

2. It is usual in acute middle ear infections, even in the early stages, to find evidences of involvement of nerve or end organ. Rarely with the present methods of diagnosis are we unable to find evidences of such involvement.

3. The lesion of nerve or end organ is not necessarily dependent upon the acute middle ear infection. There is no definite relation between the severity of the middle ear infection and the lesion of nerve or end organ. When the middle ear infection is unilateral the unaffecetd ear usually presents evidence of a lesion of nerve or end organ.

4. After the disappearance of the acute middle ear infection there may remain a permanent lesion of nerve or end organ.

5. In every case of chronic otorrhea except one there was evidence of a lesion of nerve or end organ. There is an interdependence between the middle ear lesion and that of the nerve or end organ.

6. In the H. O. M. cases every ear studied save one presented evidence of lesion of nerve or end organ.

7. In H. O. M. there is no constant relation between the extent of the lesion in the middle ear and the lesion of the nerve or end organ.

OUTLINE FOR NEUROLOGICAL, NERVE DEAFNESS AND LABYRINTHINE CASES.

NAME DATE

	Before inflation		After inflation	
Hearing (Right	W	S	W	S
(Left	W	S	W	S

Paracusis Willisii
Tinnitus
Dizziness
Staggering
Vomiting

Tests

Rinne (Right Weber Politzer Bone conduction (R
(Left (L

Fistula Test:
Gelle Test:

Hearing	50DV	C	C^1	C^2	C^3	C^4	C^5
Right							
Left							

Low limit High limit

Balance sense

Romberg-walking crack On one foot Hopping, eye shut

Tests of vestibular apparatus

Spontaneous

Past-pointing Extra ocular muscle balance
Maddox Dist. Near
Bi. Bo. Bu. Bd.

Nystagmus Up Down Right Left

Caloric

Right			Left		
After	Min.	Sec.	After	Min.	Sec.
Nature of nystagmus			Nature of nystagmus		
Amp.				Amp.	

Head back
Head forward
Past-pointing
Falling

Rotation

(Eyes closed during rotation)

Head 30 degrees forward

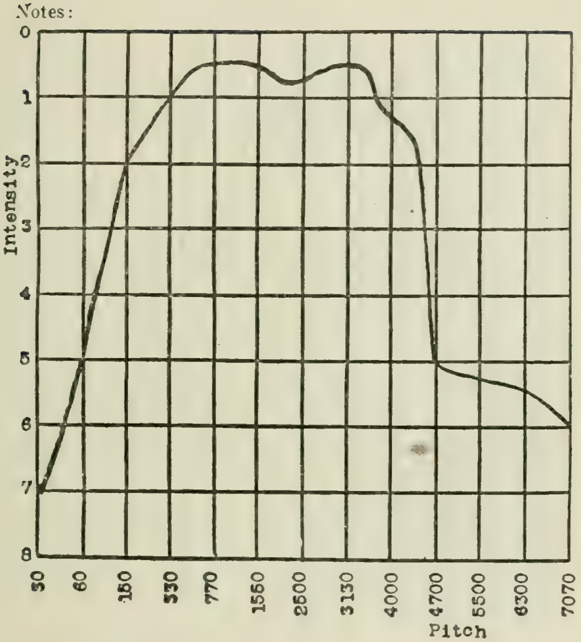
R to L turns in 20 seconds:
Nystagmus Duration
L to R turns in 20 seconds
Nystagmus Duration
R to L 10 turns in 10 seconds:
Pointing Falling
L to R 10 turns in 10 seconds
Pointing Falling

Head forward 120 degrees

R to L 10 turns in 20 seconds
Nystagmus Duration
L to R 10 turns in 20 seconds
Nystagmus Duration
R to L 10 turns in 10 seconds
Pointing Falling
L to R 10 turns in 10 seconds
Pointing Falling

Vertigo during various tests:

PLATE I.



Upper limit: Galton, R., 23,000 d. v.; Koenig, R., 43,960 v. s.
Monochord: A. C., R., 19,000 d. v.; B. C., R., 20,000 d. v.

PLATE II.

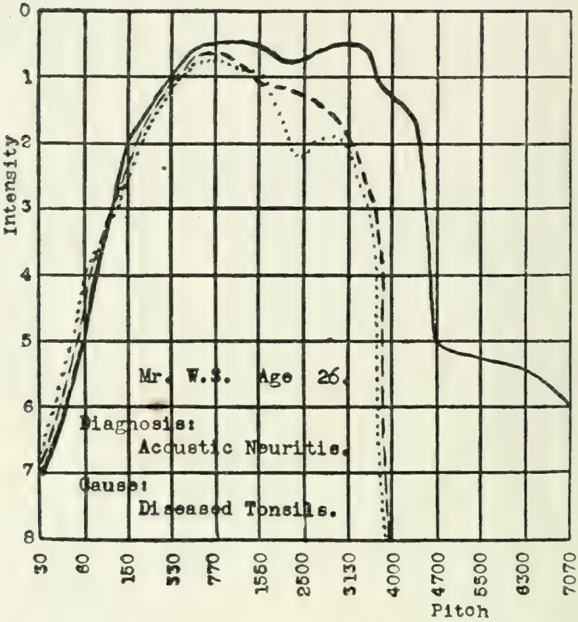
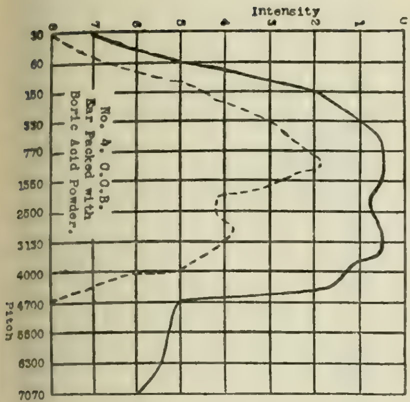
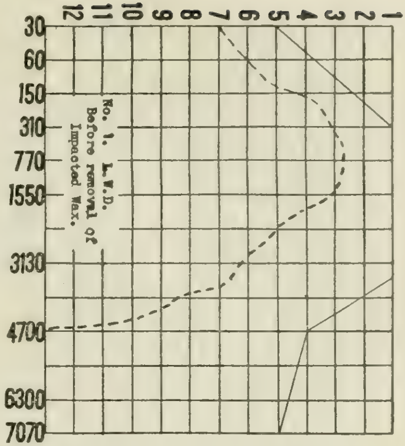

Intensity
0
1
2
3
4
5
6
7
8
30
60
150
330
770
1550
2500
3130
4000
4700
5500
6300
7070
Pitch
Mr. W.S. Age 26.
Diagnosis:
Accustic Neuritis.
Cause:
Diseased Tonsils.

PLATE III.





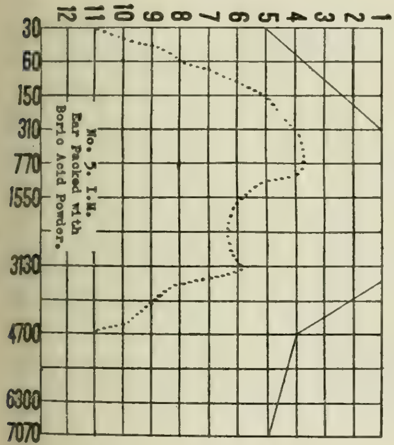
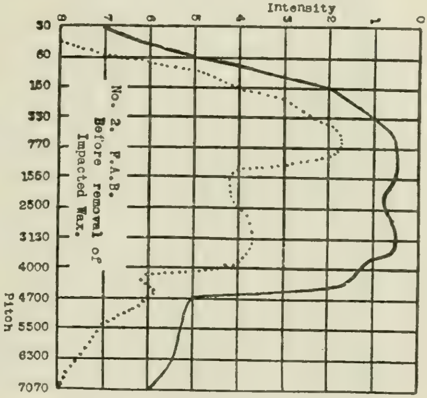
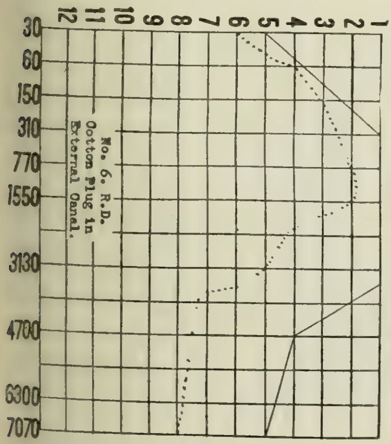


PLATE III





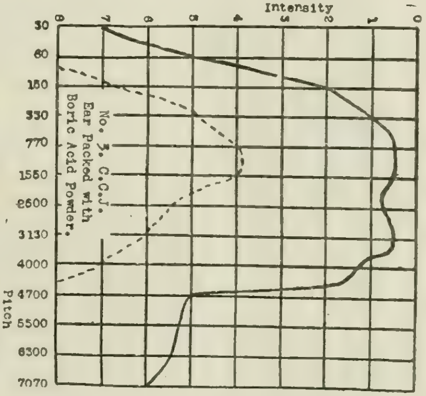
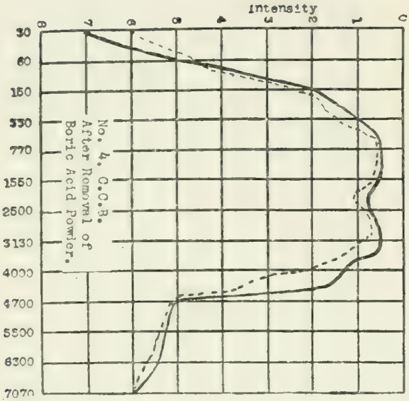


PLATE IV.



Intensity
8
7
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4000
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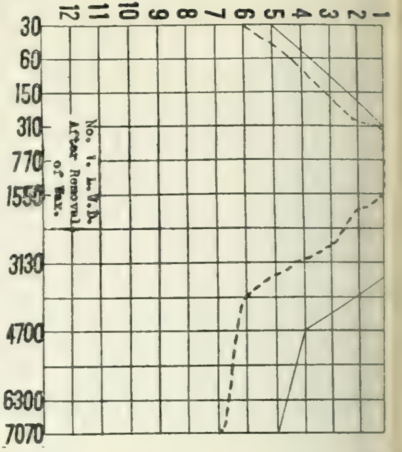
7070
No. 4. C.C.B.
After Removal of
Boric Acid Powder.



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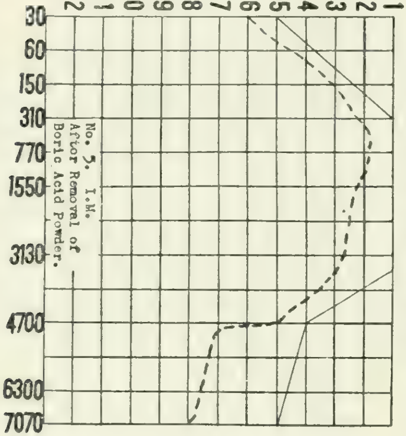
4700
6300
7070
No. 1. L.V.D.
After Removal
of Wax.



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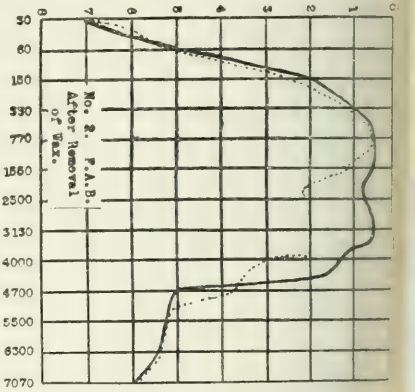
11
12
30
60
150
310
770
1550
3130
4700
6300
7070
No. 5. I.M.
After Removal of
Boric Acid Powder.



8
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150
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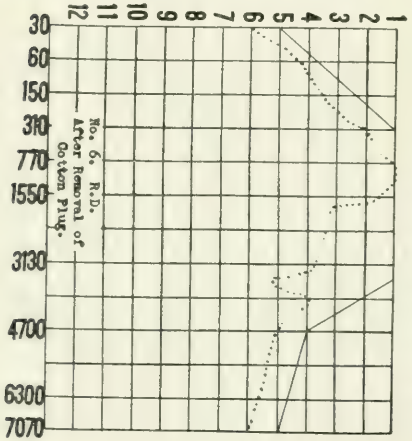
770
1550
2500
3130
4000
4700
5500
6300
7070
No. 2. P.A.B.
After Removal
of Wax.



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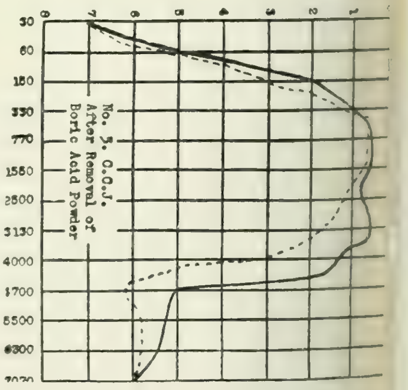
770
1550
3130
4700
6300
7070
No. 6. R.D.
After Removal of
Cotton Plug.



8
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6
5
4
3
2
1
30
60
150
330
770
1550
2500
3130
4000
4700
5500
6300
7070
No. 3. C.C.J.
After Removal of
Boric Acid Powder

PLATE V.

Upper limits in cases of occlusion of external auditory meatus.

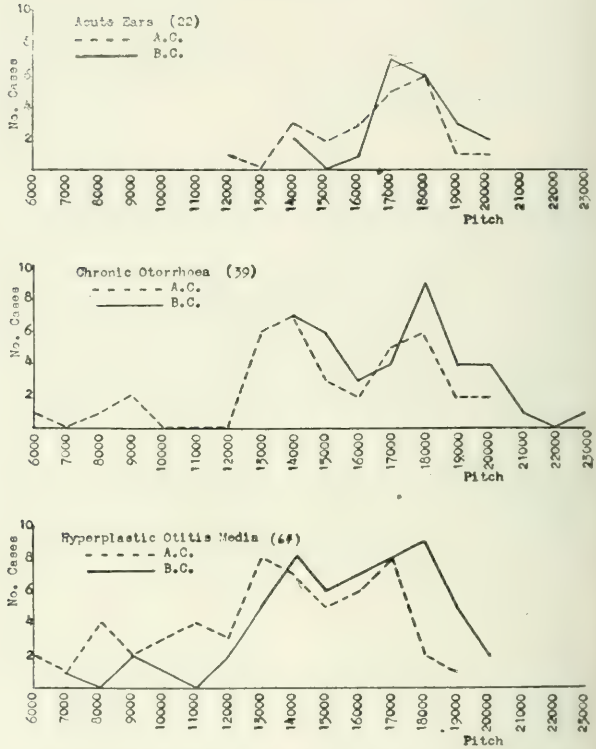
Before Removal

No. 2.	F. A. B.	A. C. 17,000	B. C. 19,000
No. 3.	C. C. J.	A. C. 15,000	B. C. 18,000
No. 4.	C. C. B.	A. C. 17,000	B. C. 20,000

After Removal.

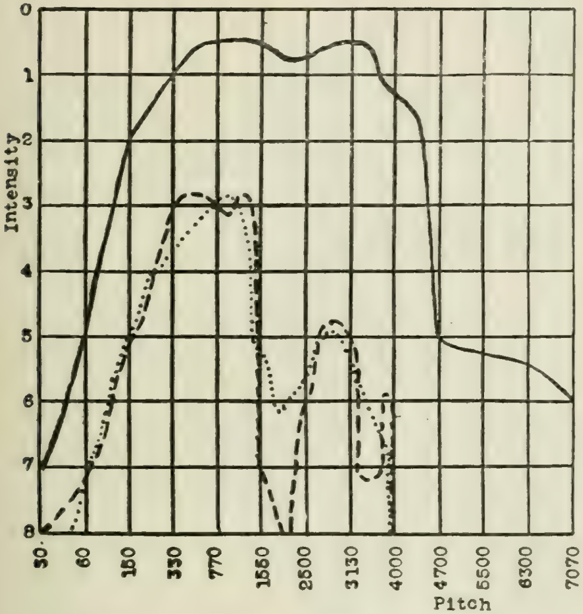
No. 2.	F. A. B.	A. C. 18,000	B. C. 19,000
No. 3.	C. C. J.	A. C. 17,000	B. C. 19,000
No. 4.	C. C. B.	A. C. 19,000	B. C. 20,000

PLATE VI.



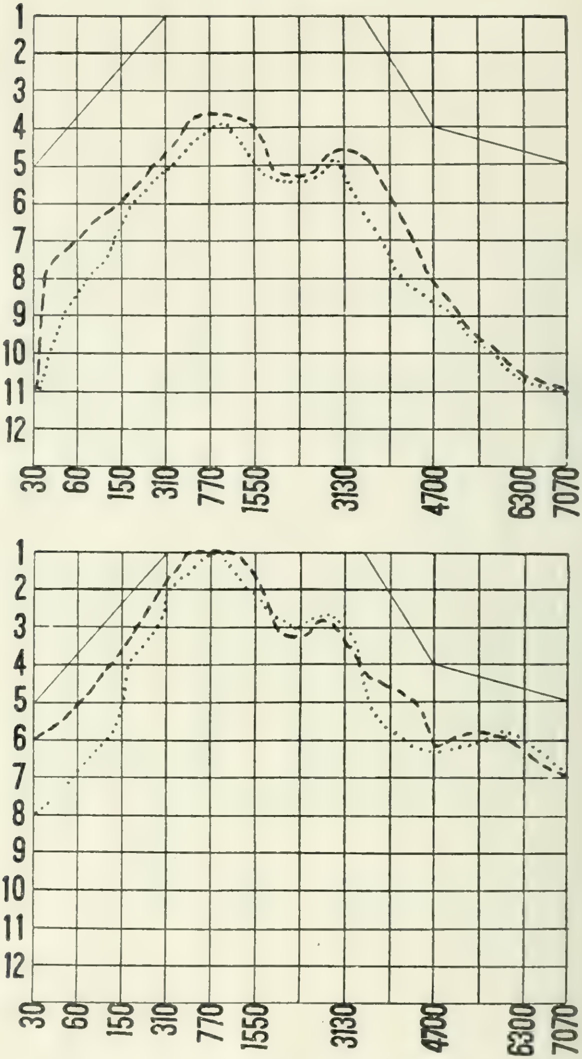
Upper limit—Comparisons of A. C. and B. C. with monochord.

PLATE VII.



Mr. N., age 17. Cause: Mumps.

PLATE VIII.



Mr. C. C., age 15. Diagnosis: Acute tubal catarrh. Before inflation: Voice, R., Wh. 9 ft., Sp. 16 ft.; L., Wh. 6 ft., Sp. 15 ft. After inflation: R., Wh. 15 ft., Sp. 45 ft.; L., Wh. 15 ft., Sp. 45 ft. Upper limit, monochord: R., A. C. 16,000, B. C. 19,000; L., A. C. 17,000, B. C. 19,000.

PLATE IX.

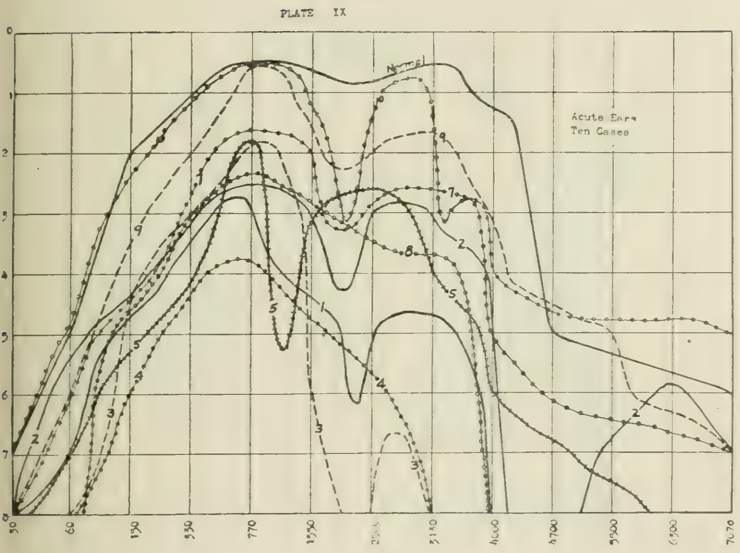
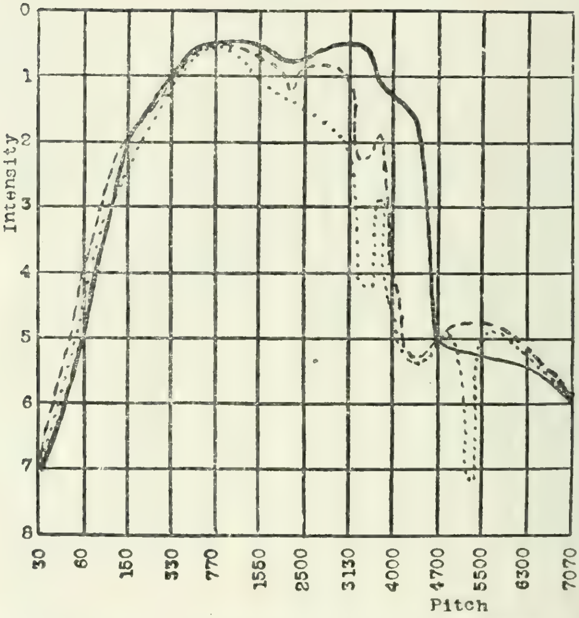


PLATE IX
Acute Ears
Ten Cases
Normal
0
1
2
3
4
5
6
7
8
30
60
130
330
770
1550
3130
4000
4700
5500
6300
7070
1
2
3
4
5
7
8
9

PLATE X.



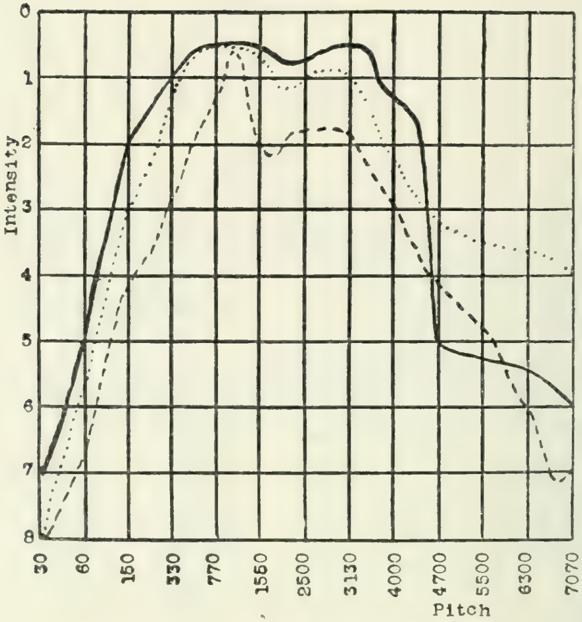
Mr. H. B., age 18. Diagnosis: Acute otitis media. (Twelve hours' duration.)

PLATE XI.

Uneffected Ears in Unilateral Cases.

Name	Rinne	B. C. (c$_2$ fork)	Monochord A. C.	Monochord B. C.
A. A. S.	+	Normal	19,000	19,000
M. K.	+	Normal	15,000	17,000
G. V. B.	+	Normal	16,000	17,000
E. C.	+	—	17,000	18,000
B. D.	+	—	18,000	18,000
E. S.	+	—	19,000	21,000

PLATE XII.

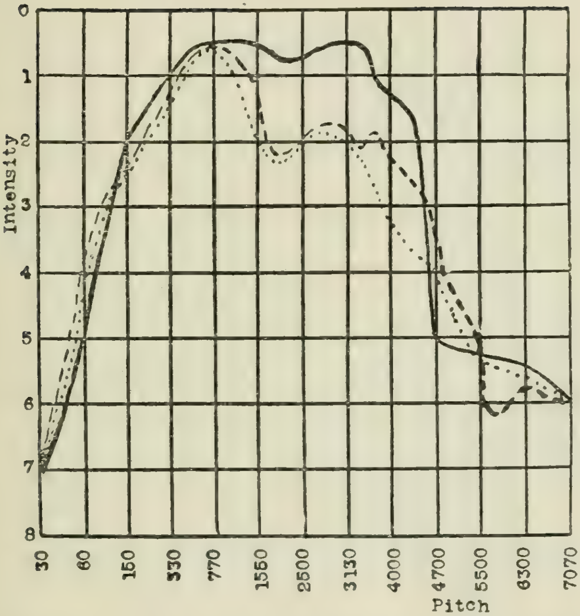


B. D., age 22. Voice, R., W. 8 ft., Sp. 40 ft.; L., W. 30 in., Sp. 36 ft. B. C. (c^2 fork), R., 1 sec.; L., 4 sec. Upper limit: Monochord—R., A. C. 18,000, B. C. 18,000; L., A. C. 18,000; B. C. 19,000.

Forks	50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R....	4/4	8/8	11/12	22/24	36/36	44/48	50/60
L....	4/4	8/8	10/12	20/24	36/36	40/48	40/60

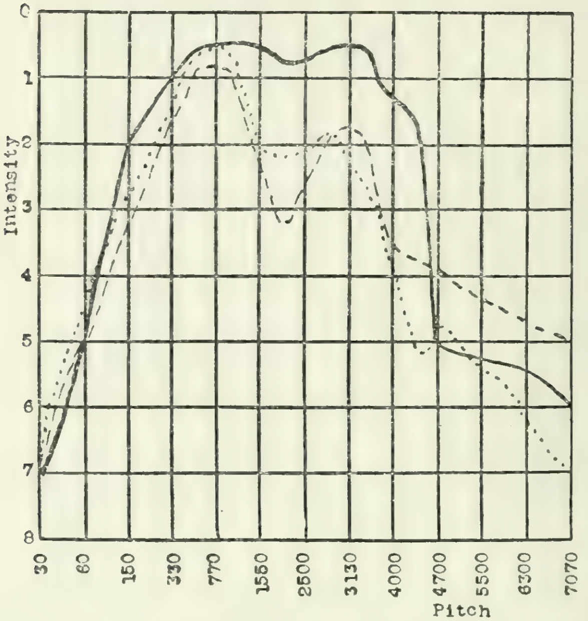
Diagnosis: Acute suppurative otitis media left.

PLATE XIII.



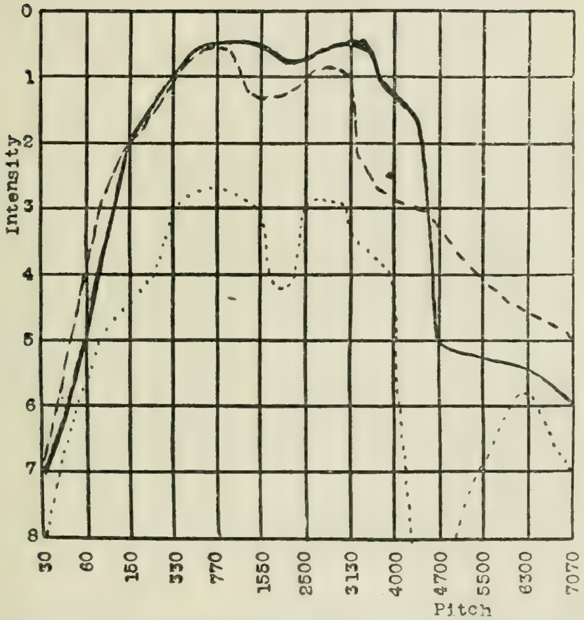
E. B., age 49. Voice: R., Wh. 12 ft., Sp. 45 ft.; L., Wh. 13 ft., Sp. 45 ft. B. C. (c^2 fork), R., —2 sec., L., —2 sec. Upper limit, monochord, R., A. C. 14,000, B. C. 17,000; L., A. C. 13,000, B. C. 17,000. Diagnosis: Acute tubal catarrh.

PLATE XIV.



E. B., age 49. B. C. (c^2 fork), R., —3 sec., L., —3 sec. Upper limit, monochord, R., A. C. 16,000, B. C. 17,000; L., A. C. 14,000; B. C., 17,000.

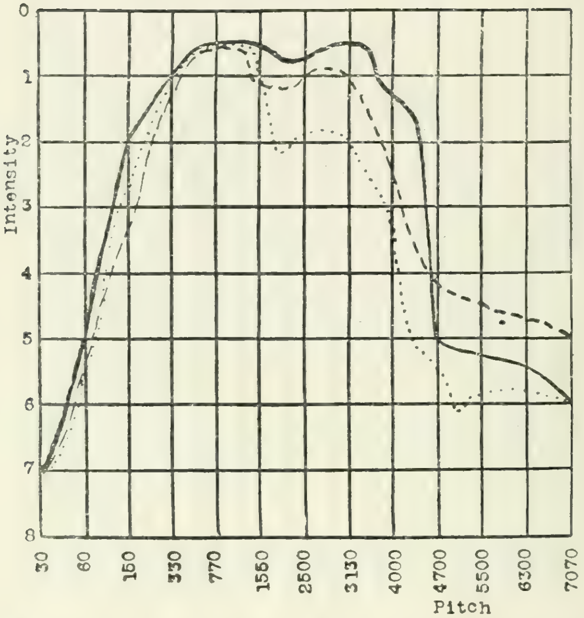
PLATE XV.



Mr. A. A. S., age 18. Voice, R., Wh. 15 ft., Sp. 45 ft.; L., Wh. 10 in., Sp. 2 ft.

Forks	50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R.....	4/4	8/8	12/12	24/24	36/36	48/48	55/60
L.....	3/4	6/8	7/12	18/24	24/36	20/48	15/60

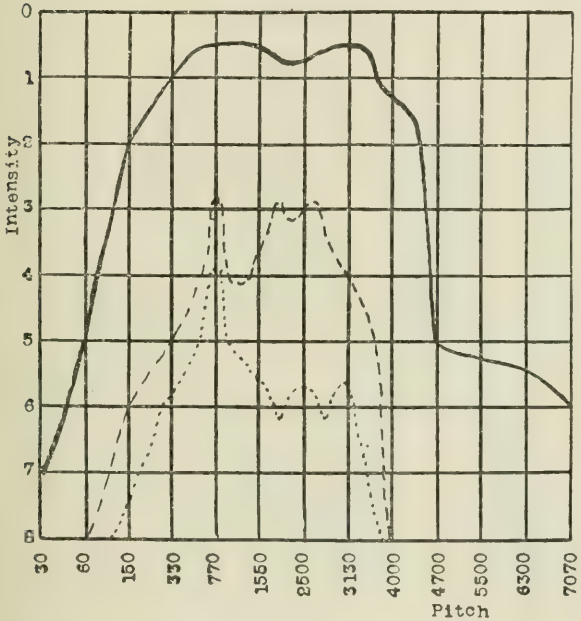
PLATE XVI.



Miss B. D., age 22. Voice, R., Wh. 13 ft., Sp. 45 ft.; L., Wh. 12 ft., Sp. 45 ft. B. C. (c^2 fork), R., —5 sec., L. —4 sec. Upper limit, monochord, R., A. C. 19,000, B. C. 19,000; L., A. C. 18,000, B. C. 19,000.

Forks	50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R.....	4/4	8/8	12/12	24/24	36/36	48/48	60/60
L.....	4/4	8/8	12/12	24/24	36/36	40/48	55/60

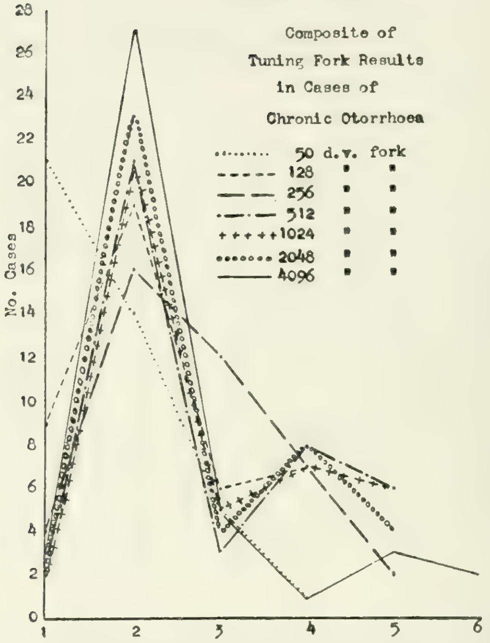
PLATE XVII.



R. B., age 14. Diagnosis: Bilateral chronic otorrhea, persisting after bilateral radical mastoid operation. Voice: R., Wh. 18 in., Sp. 3 ft.; L., Wh. 4 in., Sp. 9 in. B. C., R. and L. increased (c^2 fork). Rinne, R. and L. negative. Upper limit, monochord, R., A. C. 14,000, B. C. 15,000; L., A. C. 13,000, B. C. 15,000.

Forks	50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R.....	0	0/8	1/12	1/24	1/36	1/48	0/60
L.....	0	0/8	0/12	0/24	0/36	1/48	0/60

PLATE XVIII.



1 Not Heard
2 Heard by a fraction equivalent to or less than 1/4
3 " " " " between 1/4 and 1/2
4 " " " " " 1/2 " 3/4
5 " " " " " 3/4 " 1/1
6 Normal

PLATE XIX.

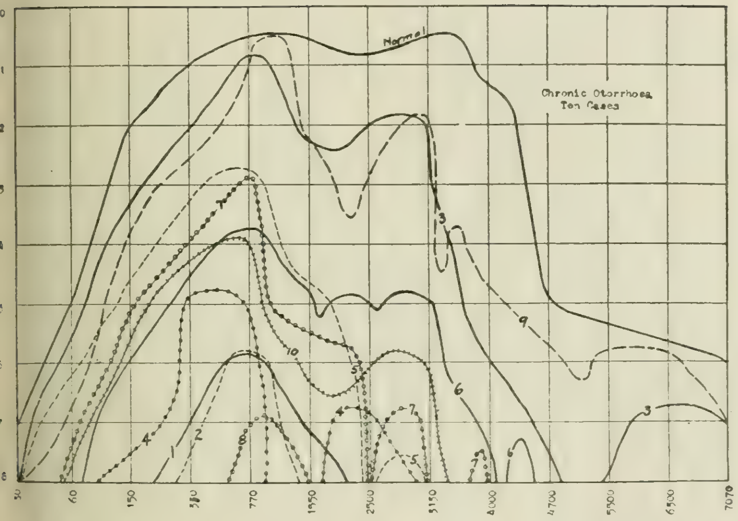
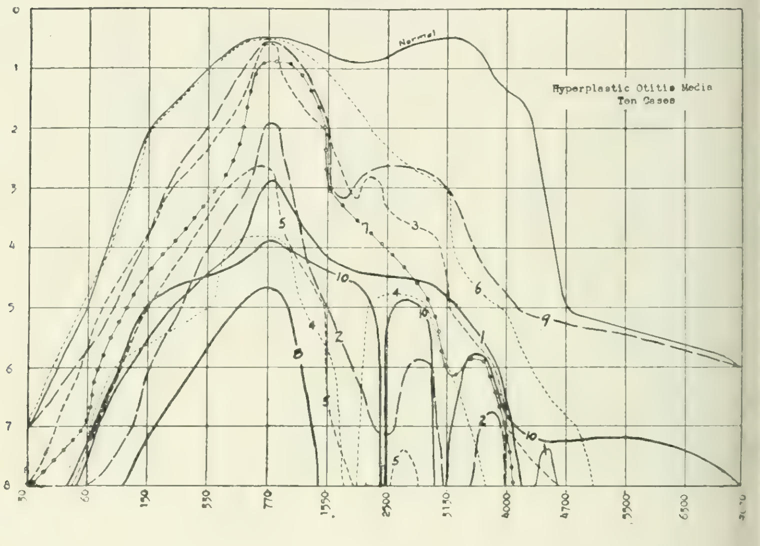

Normal
Chronic Otorrhoea
Ten Cases
1
2
3
4
5
6
7
8
9
10
0
6
30
60
150
350
770
1950
2500
3150
4000
4700
5500
6500
7070

PLATE XX.


Hyperplastic Otitis Media
Ten Cases
Normal
0
1
2
3
4
5
6
7
8
30
60
130
330
770
1550
2500
3150
4000
4700
5500
6500
1
2
3
4
5
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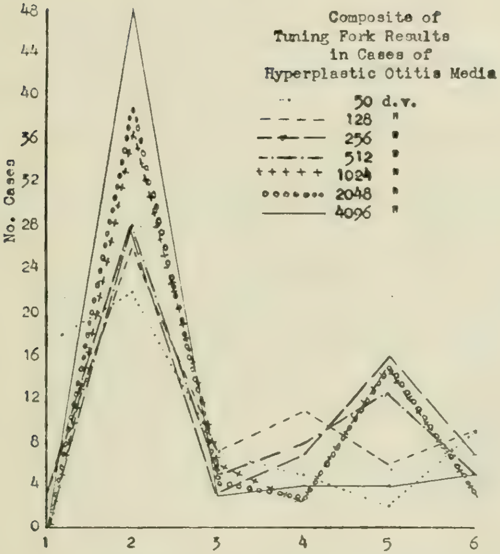
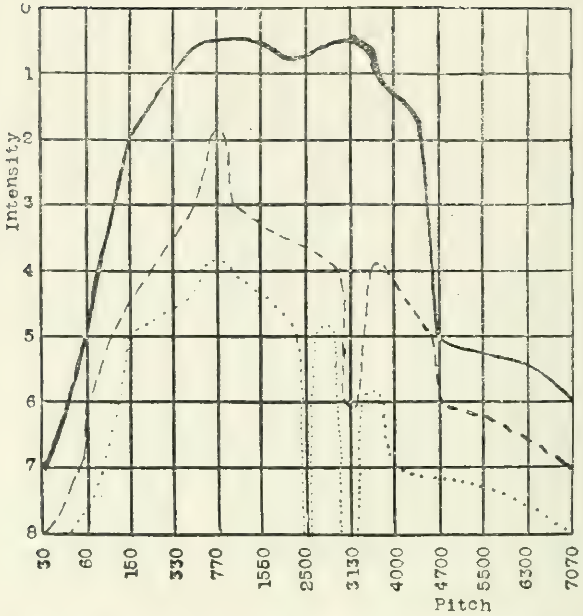
7
8
9
10

PLATE XXI.



1 Not heard
2 Heard by a fraction equivalent to or less than 1/4
3 " " " " between 1/4 and 1/2
4 " " " " " 1/2 " 3/4
5 " " " " " 3/4 " 1/1
6 Normal

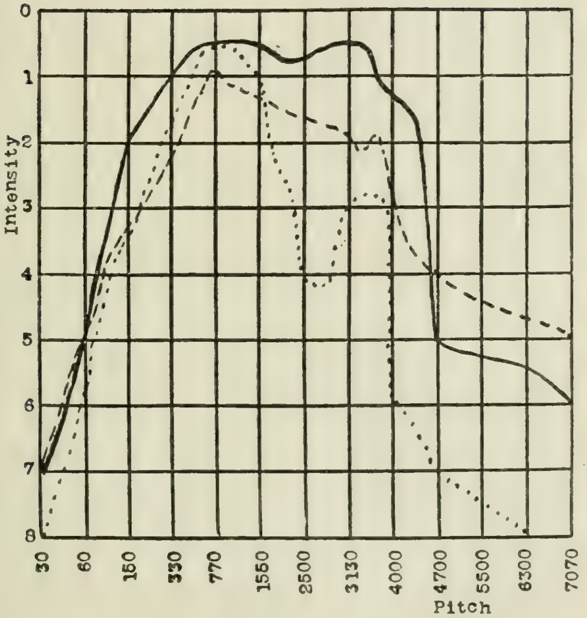
PLATE XXII.



F. K., age 27. Diagnosis: Hyperplastic otitis media. Voice: R. and L., Whisper 2 ft. Rinne: R. and L. negative. B. C. (c^2), R., —2 sec., L. —1 sec. Upper limit, monochord, R., A. C. 17,000, B. C. 17,000; L., A. C. 16,000, B. C. 17,000.

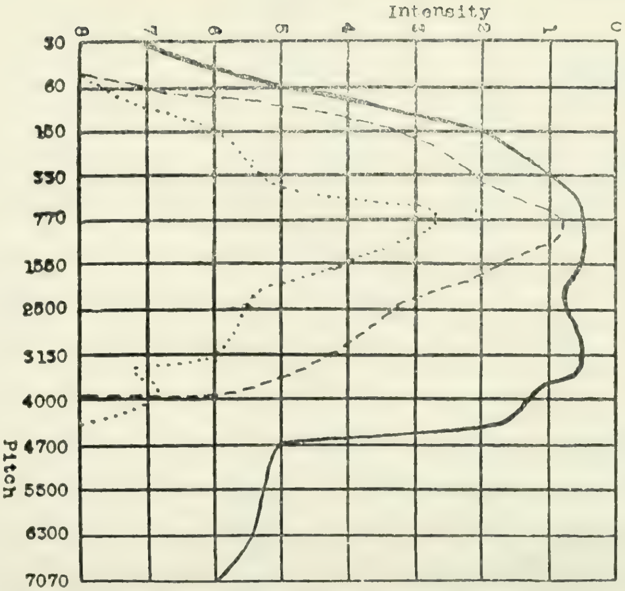
Forks	50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R.....	0	0/8	2/12	4/24	6/36	12/48	15/60
L.....	0	0/8	1/12	3/24	3/36	6/48	8/60

PLATE XXIII.

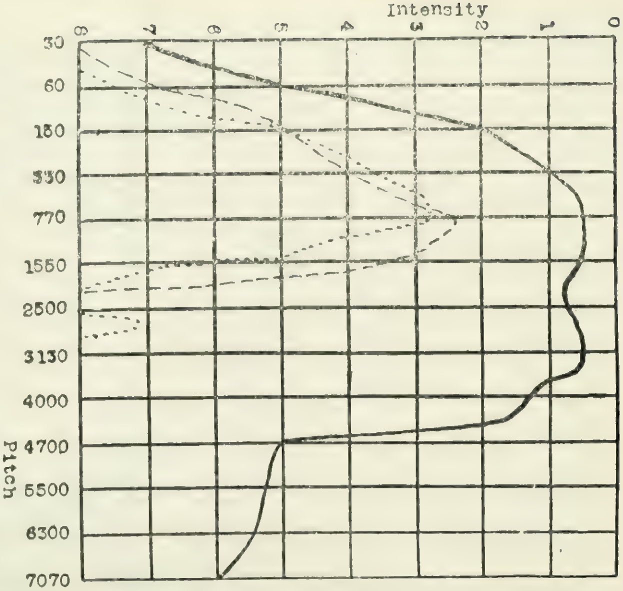


Mrs. C. J. L., age 25. Voice: R., Wh. 5 ft., Sp. 15 ft.; L., Wh. 10 ft., Sp. 30 ft. Diagnosis: Hyperplastic otitis media. B. C. (c^2), R., normal; L., —5 sec. Rinne: R. neg., L. pos. Upper limit, monochord, R., A. C. 17,000, B. C. 19,000; L., A. C. 18,000; B. C., 20,000.

PLATE XXIV.

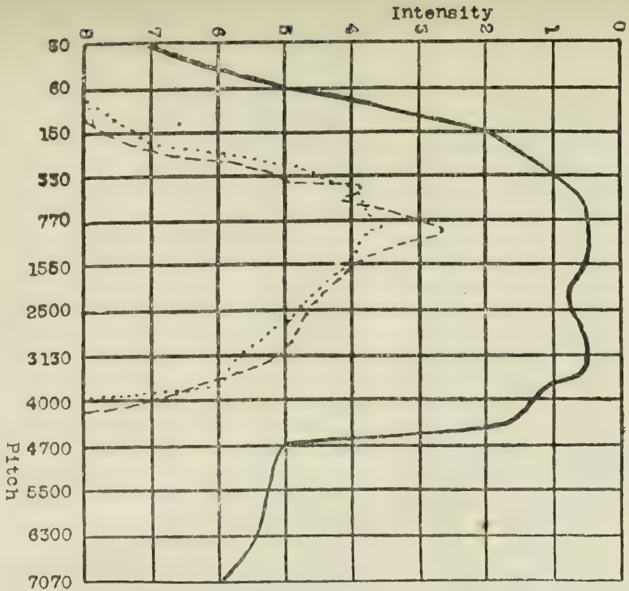


H.S., age 62. Diagnosis: Hyperplastic otitis media. Voice: R., Wh. 1 ft., Sp. 3 ft.; L., Wh. 6 in., Sp. 18 in. Rinne: Positive each ear. B. C.: R., increased 2 sec., L., decreased 5 sec. Upper limit: Monochord, not heard; Galton whistle, not heard; Koenig cylinders, R. and L., 21,845 v. s.



C. P., age 62. Diagnosis: Hyperplastic otitis media. Voice: R., Wh. 15 ft., Sp. 45 ft.; L., Wh. 6 ft., Sp. 20 ft. Rinne: Positive each ear. B. C.: R., —5 sec., L., —12 sec. Upper limit, monochord: R., A. C. 13,000, B. C. 15,000; L., A. C. 14,000, B. C. 16,000.

PLATE XXV.

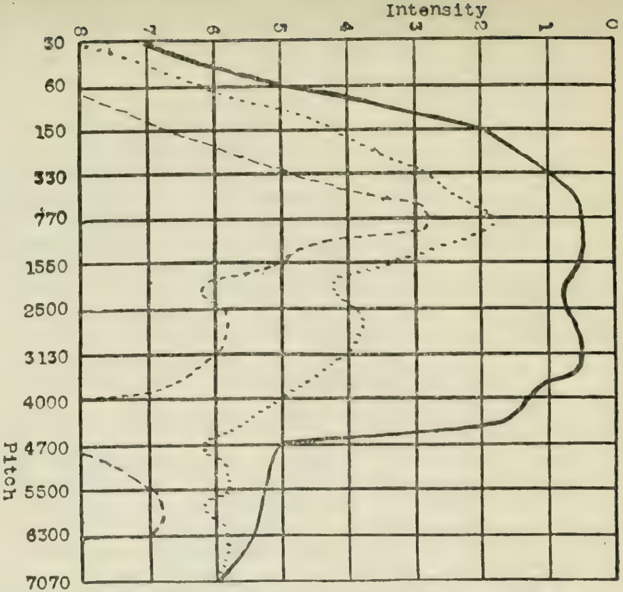


L. D., age 25. Voice: R., Wh. 6 in., Sp. 18 in.; L., Wh. 4 ft., Sp. 10 ft. B. C. (c^2), R. normal, L., increased.

Forks:

	50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R...	0	1/8	1/12	2/24	8/36	5/48	2/60
L...	2/4	6/8	8/12	10/24	20/36	24/48	30/60

Upper limit: Monochord, R., A. C. 17,000, B. C. 19,000; L., A. C. 17,000, B. C. 19,000.



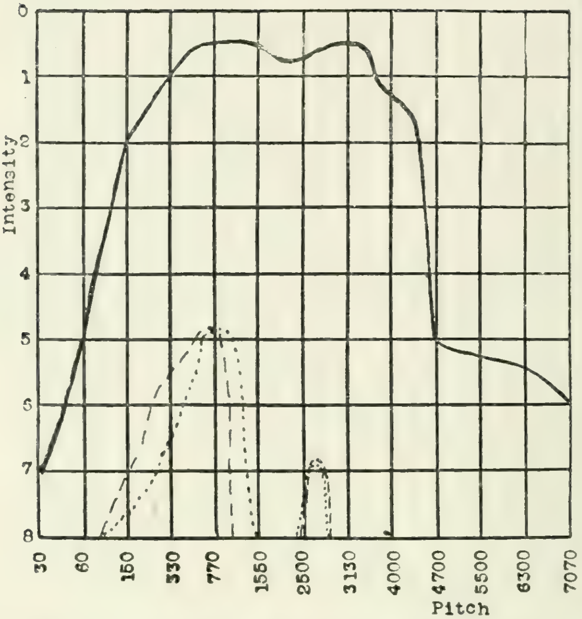
H. B., age 20. Voice: R., Wh. 2½ ft., Sp. 5 ft.; L., Wh. 2½ ft., Sp. 4 ft. B. C. (c^2), R., —4 sec., L. —6 sec.; (c^1), R., —4 sec., L. —5 sec.

Forks:

	50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R....	0	1/12	1/18	1/24	1/36	2/48	1/60
L....	0	1/12	1/18	1/24	1/36	2/48	1/60

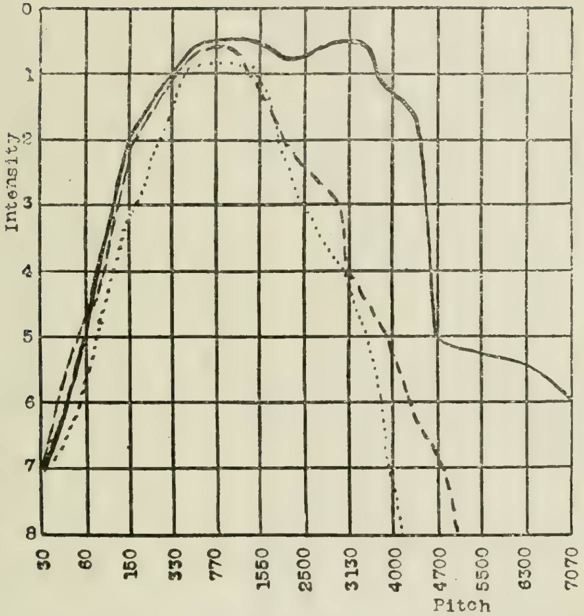
Upper limit: Monochord, R., A. C. 15,000, B. C. 17,000; L., A. C. 13,000, B. C. 15,000.

XXVI.



H. J., age 21. Diagnosis: Hyperplastic otitis media; Wassermann, 4 plus. Voice: R. Wh. 0, Sp. 11 in.; L., Wh. 0, Sp. 11 in. Rinne: c^2 positive on each side; c^1 negative on each side. B. C. (c^2) —14 sec. each side; (c^1) —6 sec. each side. Upper limit: Monochord, R., A. C. 8,000, B. C. 9,000; L., A. C. 8,000; B. C. 9,000.

PLATE XXVII.



J. C., age 48. Voice: R., Wh. 9 ft., Sp. 45 ft.; L., Wh. 8 ft., Sp. 40 ft. Rinne: Positive each side. B. C. (c^2), R., —6 sec., L. —4 sec.

Forks	50 d. v.	c	c^1	c^2	c^3	c^4	c^5
R...	4/4	7/8	10/12	20/24	30/36	40/48	0/60
L...	4/4	8/8	12/12	24/24	30/36	40/48	0/60

Upper limit: Monochord, R., A. C. 11,000, B. C. 12,000; L., A. C. 12,000, B. C. 13,000. Diagnosis: Hyperplastic otitis media.

XL.

OBSERVATIONS ON HAY FEVER.

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The symptom complex to which John Bostock in 1819 gave the name hay fever may attack an individual at various seasons of the year, or may be present more or less continuously throughout the entire year. The former is called seasonal hay fever, and the latter is called perennial hay fever. Seasonal hay fever is a very common complaint, and it has been estimated that one per cent of the population of the United States is so afflicted. Perennial hay fever is much less common. However, if it is proper to include those cases who are subject to frequently repeated head colds and those who have vasomotor rhinitis, both of which conditions are not very unlike perennial hay fever, it then becomes a very frequent complaint.

PERENNIAL HAY FEVER.

Perennial or all the year hay fever is very often caused by the inhalation of the proteins of the hair, dandruff or skin dust of the horse, dog, cat and other fur bearing animals, such as pets and fur wearing apparel. The same holds true for the feathers of the chicken and goose, for example, feather pillows, and feathered pets, such as parrots. The inhalation of plant pollens at a definite season may predispose to a perennial hay fever, and a seasonal hay fever, caused by pollens, such as timothy, grass and ragweed, may become an all the year condition due to other superimposed causes. The inhalation of the flour of the cereal grains is not infrequently a cause of perennial hay fever; such instances are confined to bakers, cooks and those who handle the various types of flour and ground up grain. In fact, the inhalation of any kind of organic dust may cause perennial hay fever; instances of this are the inhalation of face powders and of boxwood and organe wood dust by jewel polishers, etc. Therefore the patient's occupation, habits and surroundings may have a bearing on the cause of the symptoms in this inhalation type of perennial hay fever.

Another group of causes of perennial hay fever we designate as the ingestion type, and this concerns the eating of food proteins. Although in my experience eggs, wheat and milk are the usual food proteins that are found to cause this condition, tests should be made with all of the common foods that the patient is continuously or frequently in the habit of eating. Among the common foods that seem essential to test routinely are egg, wheat, milk, rice, corn, oat, potato, pea, bean, beef, lamb, pork and chicken; additional foods may be determined from the patient. Examples of unusual foods causing perennial hay fever follow: A woman had hay fever from eating raw carrots which she craved and ate every day; another had hay fever whenever she ate celery, and a third patient noticed that among other foods onions caused hay fever.

The above mentioned two types of causes of perennial hay fever, namely, the inhalation and the ingestion types, may be determined by means of skin or cutaneous tests, the technic of which follows. A number of small cuts, each about an eighth of an inch long, are made on the flexor surfaces of the forearm. These cuts are made with a sharp scalpel, but are not deep enough to draw blood, although they do penetrate the skin. On each cut is placed a protein and to it is added a drop of tenth normal sodium hydroxid solution to dissolve the protein and to permit of its rapid absorption. In the case of pollens, animal hair and feathers a concentrated solution of pollen protein or animal hair extract may be used without the addition of sodium hydroxid. At the end of half an hour the proteins are washed off and the reactions are noted, always comparing the inoculated cuts with normal controls on which no pollen was placed. A positive reaction consists of a raised white elevation or uticarial wheal surrounding the cut. The smallest reaction that we consider positive must measure 0.5 cm. in diameter.

The treatment of the above mentioned two types of perennial hay fever, namely, the inhalation and ingestion types, consists chiefly in the omission of the offending protein as determined by the positive skin test. Therefore, treatment may consist of the omission of a food from the diet or the avoidance of domestic animals and pets, or discarding the feather pillows, or a change in occupation, or discontinuing the use of a certain face powder, etc. Since the avoidance of exposure to animals is not always possible and the avoidance of exposure to common pollens is rarely possible, desensitizing treatment may be given for the former and should be given for the latter. This method of treatment will be described later on, when we discuss seasonal hay fever.

A third type of perennial hay fever we call infectious type because bacteria seem to be the prominent cause. This type is determined by the process of exclusion, and it comprises those patients who not only fail to give a positive cutaneous test but also have no demonstrable rhinologic cause. In other words, cutaneous tests and rhinologic examination have failed to determine the cause of symptoms. For the treatment of this infectious type of perennial hay fever we have to resort to vaccines. Although stock vaccines sometimes give good results, it is best to use autogenous nasal vaccines which are prepared by inoculating the patient's nasal secretion either into dextrose bouillon or into blood agar plates. Vaccine treatment should not be given oftener than at five day intervals and preferably only at weekly intervals, and the dose of vaccine should be gradually increased.

A fourth type of perennial hay fever has already been casually mentioned and concerns the rhinologist. Among the various causes may be mentioned the presence of polyps, adenoids, sinus infection, etc. The rhinologist will determine the cause and treatment of this type; when, however, he fails, autogenous nasal vaccines are worth trying and often benefit.

Dr. Goodale has called attention to a pseudohay fever due to the fragrance of certain heavily scented flowers. It seems to me that this pseudo type of hay fever is due to many causes which may be classified as mechanical, chemical, odorific and thermal. Among the mechanical causes any kind of dust is the most frequent cause, more especially sweeping dust and hay dust; fine powder, such as talcum and the like, is also a frequent cause. Among the chemical irritants, soap powder, lye and ammoniac fumes are very frequent causes. Among the odorific irritants, heavily scented perfumes, face powders, musty air and stable odors are frequent causes. Thermal irritants concern sudden changes of temperature, as in going from warm air to extreme cold, from moist air to very dry air, and exposure to drafts; a very frequent history is that of a paroxysm of sneezing, with or without running of the nose, on retiring and on arising. The mechanism seems to be a reflex due to the sudden exposure of the warm and protected skin of the body to cold air, as in getting out of bed and in undressing, during which acts the warm body surface is suddenly and momentarily exposed to cool air; in other words, there is a mild chilling of the body surface. The same mechanism holds for many who take cold easily. Some of these patients are sensitive to some type of protein which may have rendered their nasal mucous membranes sensitive to these irritants; others are not sensitive to proteins. Appropriate protein treatment for those who are sensitive usually relieves these symptoms, no matter what the irritant may be, and occasionally autogenous nasal vaccines will benefit or relieve the nonsensitive individual.

Therefore, to summarize, I think that the following procedures offer the best results in the determination of the cause and treatment of perennial hay fever: First do routine cutaneous tests with the common food proteins, common pollen proteins, and domestic animal hair proteins; the need of testing with unusual food proteins, face powders and other unusual types of proteins depends upon the customs and habits of the individual in question. A positive test followed by omission of the protein yields satisfactory results.

With those patients in whom cutaneous tests fail to determine the cause, an examination of the nasal cavities and sinuses may reveal the cause. Autogenous nasal vaccines often have to be given even though previous examinations and tests have or have not revealed a cause. The same procedure of testing, rhinologic examination and vaccine therapy may be applied to the condition of frequent colds. With the vasomotor rhinitis cases I should put rhinologic examination first, then if necessary cutaneous tests and autogenous vaccines. The possibility of a pseudohay fever must be borne in mind, and this may be a primary condition or secondary to other causes.

SEASONAL HAY FEVER.

For convenience we classify or divide seasonal hay fever into three groups, namely, spring, summer and autumn. The spring type concerns those who have symptoms during February, March, April and May, and the causative agent is usually the pollens of trees. The summer type concerns those who have symptoms during late May, June and July, and the causative agent is usually the pollens of grasses. The autumn type concerns those who have symptoms during August and September, and the causative agent is usually the pollen of ragweed; dwarf ragweed in the East and giant ragweed in the West. Naturally the season of pollination of these various plants may vary according to the locations. However, the history of the patient will tell you the time of year when symptoms are present, and it is up to you to determine the prevailing causative pollen. What I shall tell you applies to the New England states, and this will give you a working knowledge for your respective localities. In fact, I am inclined to believe that the causative pollens do not vary very much for different localities, but the time of pollination of the same plant does vary in different localities.

HAY FEVER CAUSED BY THE POLLEN OF TREES.

The first pollen season begins in February and continues into May, during which time various trees pollinate. Silver maple often pollinates in February; the other maples, birches, willows and hazel nut pollinate in late March or early April; the poplars, juniper, cottonwood and elm pollinate during April; the oaks, ash, bayberry and hickory pollinate during May; the pines pollinate in late May and early June, and the fruit trees pollinate in May. I have observed twelve patients who were sensitive to and had hay fever from the pollen of trees. One patient had hay fever caused by the pollen of apple blossoms, and he was free from symptoms following preseasonal treatment with its pollen extract. One patient, who was sensitive to the pollen of the oak and maple, and another, who was sensitive to the pollen of the willow, were both free from symptoms following preseasonal treatment with these pollen extracts. Patients not treated were those sensitive to the following tree pollens: one patient, to poplar tree pollen; one, to pine tree pollen; two, to ash; one, to willow; two, to both willow and poplar, and one to willow, poplar and ash. Since the season of pollination of the individual trees continues only from a few days to two weeks at the most, it does not seem essential that treatment be given. However, treatment may be successfully given for tree pollens in the same manner as for other pollens for which treatment will be detailed later on.

CAUSES OF THE SUMMER TYPE OF HAY FEVER.

Patients who have hay fever during late May, June and July, the so-called rose cold period, are exposed to many kinds of pollens, but actually the pollens of only the grasses cause much trouble. In the New England States some of the compositæ, the most important of which is daisy, pollinate at this time, but for reasons given later on daisy pollen rarely, if ever, is a cause of hay fever. Such plants as the lilies, sorrel, buttercup and others pollinate at this time, but when tested no patient has been found to be sensitive to these, and if they ever cause hay fever it is because of immediate nasal contact with the blossom. The rose which pollinates at this time is rarely a cause of hay fever, since it is not a wind pollinated plant. Since, however, rose is commonly suspected to be the cause of hay fever or rose colds, and since the pollen of rose is occasionally the actual cause, skin tests with rose-pollen frequently have to be done, if for no other reason than to satisfy the patient. So far I have had reason to treat only three rose cases.

The cause of hay fever during June and July in the New England States is practically limited to the pollens of the grass family. Lawn grass begins to pollinate early in May, as a rule, but since it is rare for hay fever to begin early in May, lawn grass pollen is probably rarely, if ever, the chief cause of hay fever. Since, however, lawn grass continues to pollinate throughout the summer, even when it is repeatedly mowed closely, with those patients who are sensitive to the pollens of other grasses and are either not treated or are insufficiently treated with the particular grass pollen that is the chief cause of hay fever, exposure to lawn grass pollen may be a complicating cause or may aggravate the symptoms of hay fever. Orchard grass pollinates during July, but this type of grass grows in more or less secluded places, and it is not commonly encountered. I have not tested a patient who reacted strongly to orchard grass. Since corn is a member of the grass family, and since the table variety pollinates during July, it must be considered among the possible causes of early hay fever. Corn pollen is very heavy. It rapidly descends to the ground and is carried by air currents or wind only a few feet. Therefore, intimate exposure would be required to produce symptoms. Furthermore, only rarely would a person be unable to avoid it. Therefore, corn pollen must rarely, if ever, be considered a cause of hay fever in the New England States, where no large acreage exists in any locality as is the case in the West. This same statement is, likewise, true of wheat, oats, barley and rye.

The grasses, then, with which we are concerned in New England are June grass, timothy and redtop, the pollens of which are light and are carried by wind considerable distances. June grass begins to pollinate some years as early as the middle of May; when the season is very late it does not pollinate before the last day or two in May; pollination continues for about three weeks. Timothy and redtop begin to pollinate between the middle of June and the first of July, depending on the season, and pollination continues until the middle or last of July; usually, the season of pollination lasts about six weeks. Therefore, in order to complete the schedule of treatment, mentioned later on in this paper as being desirable, preseasonal treatment with June grass must begin the first of March at the latest, and with timothy and redtop it must begin the middle of March.

CAUSES OF AUTUMNAL OR LATE HAY FEVER.

In the New England States ragweed (ambrosia artemisifolia, or dwarf ragweed) is not the only plant that pollinates during August and September, nor is it the only pollen to which individuals are exposed or with which patients should be tested. Most of the compositæ, such as golden rod, sunflower, golden glow and aster, pollinate during August and September; daisy pollinates during June and July, and dandelion in the early spring; giant ragweed is rarely encountered in the New England States. In my experience, however, pollens other than ragweed rarely, if ever, are the chief cause of symptoms during the late hay fever season. I have tested one hundred and seventy patients with the pollens of daisy, golden rod, sunflower and golden glow. Fifty per cent of those tested with these pollens gave a positive reaction with the whole pollen, but none of these gave a reaction with a 1:100 dilution of the pollen; the other 50 per cent failed to react at all with the whole pollen. Therefore, it may safely be stated that the pollens of golden glow sunflower, daisy and golden rod are not primarily causes of hay fever in New England. It should be borne in mind that daisy pollinates during June and July and has finished pollination before ragweed begins, therefore daisy cannot complicate the causes of August and September hay fever. In comparison with ragweed, in only 3.25 per cent of my cases could golden rod pollen be assumed as a possible cause of hay fever. From the investigations of Scheppegrell and others we know that the pollens of golden rod, sunflower, golden glow, daisy and aster are not carried by the wind, and that their pollens are heavy and shed very sparingly, so that for these reasons patients are not exposed to these pollens unless the flowers are kept in the house or unless sensitive patients smell of the flowers; naturally, the patient can refrain from such intimate contact and need not be treated for these.

I have outlined the pollens that may be the possible cause of hay fever at various seasons and I have also described the cutaneous test which when used with these pollens will determine the probable cause. Before treatment may be given it is essential to do cutaneous tests with various dilutions of the pollens or pollen protein in order to determine to which pollen of several the patient is most sensitive and with which pollen the patient should be treated, and with what dilution of pollen that gives a reaction on the skin, but treatment should be begun with the strongest solution that fails to give any reaction whatever.

These solutions I make as follows: To 0.5 gm. of the dry pollen is added 44 cc. of sterile physiologic sodium chlorid solution; the mixture is shaken thoroughly at frequent intervals for twenty-four hours, after which enough absolute alcohol (6 cc.) is added to the mixture to make the alcoholic content 12 per cent. Again, the mixture is thoroughly shaken at frequent intervals for twenty-four hours, after which it is centrifugalized at high speed and the supernatant fluid is pipetted off and saved. This supernatant fluid, therefore, consists of the pollen protein dissolved in a 12 per cent alcoholic physiologic sodium chlorid solution and it represents, by weight, 1 part pollen to 100 parts solvent. This 1:100 solution is used as stock, and from it other dilutions, 1:500, 1:1,000, 1:5,000 and 1:10,000 are made, using a 12 per cent alcoholic physiologic sodium chlorid solution as a dilutent. These solutions are used not only for the skin tests but for treatment, and with the addition of a small crystal of thymol they keep for many months in a cool place.

METHOD OF TREATING PRESEASONALLY WITH POLLEN EXTRACTS.

The first treatment consists of from 0.1 to 0.2 cc. of that dilution next higher than the one which gave a positive skin test, or, in other words, the first dose is 0.1 cc. or 0.2 cc. of the strongest dilution which failed to give any skin reaction whatever, no matter how slight. With my pollen extracts the majority of patients whom we treated gave a more or less positive reaction with the 1:10,000 dilution, therefore, the first treatment consisted of 0.1 cc. or 0.2 cc. of the 1:20,000 dilution. Treatments are given subcutaneously once a week, and each week the amount of the extract is gradually increased, so that as the treatment progressed, stronger and stronger dilutions were used, until one or more doses of the 1:100 dilution were given. As an example, I will give what I have found by experimentation to be the best outline for a patient who gives a more or less positive skin test with a 1:5,000 dilution of pollen extract; 1:10,000 give 0.15 cc.; 1:5,000 give 0.15 cc., 0.25 cc., 0.35 cc., 0.45 cc., 1:1,000 give 0.15 cc., 0.25 cc.; 1:500 give 0.15 cc., 0.25 cc., 0.35 cc., 0.45 cc.; 1:100 give 0.15 cc., 0.2 cc., 0.25 cc. Each dose was given preferably at weekly intervals and never oftener than once every five days.

This schedule of treatment calls for fourteen inoculations. However, for some reason or other, modifications frequently have to be used. Often a patient is so sensitive to the pollen that a 1:10,000 dilution gives a slight reaction, thus necessitating an initial dose of 0.15 cc. of 1:20,000 followed by possibly two doses of 1:10,000. Often it happens that a patient has considerable local or general reaction following some one treatment in the schedule, thus necessitating the repetition of that particular dose before the next increase may be given. More often the patient presents himself for treatment too late to complete the scheduled series of treatments before the onset of pollination so that for preseasonal treatment alone, some of the final treatments in the schedule must be omitted. This schedule is often modified purposely with certain individual cases. For instance, in some cases the second treatment with the 1:1,000 dilution, namely, 0.25 cc., is omitted, and in some cases instead of giving 0.15 cc. of the 1:100 dilution, when this happens to be the final treatment that the patient is to receive because of onset of pollination, a fifth treatment with the 1:500 dilution, namely, 0.55 cc., is often substituted, and even a sixth treatment with the 1:500 dilution, namely, 0.65 cc., is sometimes given. These larger doses of 1:500 approximate the amount of protein in 0.15 cc. and 0.2 cc. of the 1:100 dilution, therefore, the fifth and sixth treatment with the 1:500 dilution, as outlined, is practically the equivalent of giving 0.15 cc. and 0.2 cc. of the 1:100 dilution. Since by far the great majority of patients are treated from three to five times with the 1:500 dilution, and since this number of treatments has given fairly satisfactory results, I consider this number of treatments, which consists usually of a total of ten, as worth giving, although a continuance of the schedule beyond three doses of the 1:500 dilution is most desirable, and giving less than three treatments with the 1:500 dilution is undesirable.

Since in the New England States the majority of autumnal hay fever patients have their first symptoms between August 10 and 20, during which time the compositae, chiefly ragweed, begin to pollinate, in order to complete the above schedule just previous to the onset of symptoms and pollination patients must begin treatment between the last week in April and the first two weeks in May. Beginning treatment the first week in June permits of giving from three to five treatments with the 1:500 dilution. Likewise, since the early type of hay fever, or socalled rose cold, which is usually caused by the grasses, begins the last few days of May or the first of June, treatment for this type of hay fever should begin about the first of March, and the starting of treatment as late as the first of April will not permit of more than three to four treatments with the 1:500 dilution, according to the schedule outlined. Naturally, in localities outside of New England, these seasons would differ, and the beginning of treatment consequently would vary, also the causative pollen must differ.

My conclusions, based on a four years' experience with the treatment of June and July hay fever, are: While the pollen of timothy grass is the chief cause of hay fever, and sufficient treatment with it alone gives excellent results, it is necessary to test all patients with the pollens of redtop, June grass and rose, since an occasional patient is more sensitive to one of these pollens than to timothy pollen, and, therefore, such a patient may need treatment with one of them rather than with timothy pollen. When the same patient, as is frequently the case, is equally sensitive to the dilutions of the pollen extracts of timothy, redtop and June grass, it is advisable to treat the patient with timothy pollen extract alone because there is sufficient evidence that treatment with timothy pollen will protect against redtop pollen exposure, provided one or more treatments are given with the 1:100 dilution of the pollen extract; timothy and redtop pollinate at the same time, and redtop is less prevalent than timothy. Sufficient treatment with timothy pollen extract seems to protect also against June grass pollen exposure, but since, as is often the case, June grass pollination is well advanced or even sometimes completed before timothy pollination begins, the patients being treated with timothy have not had sufficient treatment with timothy pollen to protect them completely against June grass pollen, consequently some of these patients will have more or less symptoms from June grass exposure. Treatment with both timothy pollen and June grass pollen extracts together will not only be insufficient to protect against early June grass pollination, but the combination of the two pollen extracts necessarily retards and diminishes the amount of treatment that might be given with timothy alone. Therefore, I am opposed to treatment with pollen mixtures, except on rare occasions, and in the case of June grass it is better to take a chance on a late season with timothy protection or even an early season with few symptoms than to treat with it in conjunction with timothy when timothy treatment is essential. Rarely one meets with a patient who has hay fever only from early in May to the middle of June; in such a case treatment with June grass alone is advisable and usually sufficient. Rarely it is necessary to treat with rose pollen or some other grass than those mentioned. However, the cutaneous tests will determine this for you.

I have treated, according to the scheme already outlined, 115 grass cases of hay fever and the results from the first year's treatment follow: Twenty-eight and seven-tenths per cent were free from symptoms, 21.7 per cent were practically free, 23.5 per cent were 75 per cent benefitted, 21.7 per cent were 50 per cent benefitted and 4.3 per cent were not benefitted. Therefore, from the first year's treatment given preseasonally for grass hay fever, you may expect equally good results. Treatment of the same patient in successive years yields even better results, therefore, you can give your patients a better prognosis the second year than the first, provided as much or more treatment is given the second year as was given the first year.

My conclusions, based on four years' experience with the treatment of August and September hay fever, are that ragweed is practically the only pollen with which treatment is necessary; dwarf ragweed in the East and either dwarf or giant as the case may test in the West. In various localities there may be some pollen other than ragweed that is a causative factor. I have treated 420 cases with ragweed pollen with the following results for the first year's treatment: Twenty-one and five-tenths per cent were free from symptoms, 17.5 per cent were practically free, 25.5 per cent were 75 per cent benefitted, 29 per cent were 50 per cent benefitted, 6.5 per cent were not benefitted. Therefore, from the first year's treatment you may expect equally as good results and from successive years' treatment you may expect even better results.

The permanency of benefit from treatment seems to depend largely upon the individual and to some extent upon a large amount of treatment which renders the patient non-sensitive.

After two or three years of consecutive treatment the majority of patients will continue free or practically free from symptoms for another two or three years without treatment. After this period of time symptoms return more or less gradually. Occasionally a patient will be free only one year before symptoms return and occasionally symptoms will be as severe the first year that treatment is omitted. Rarely one season's treatment will protect for several years and I think treatment every other year with some individuals will keep the patient quite free from symptoms. As a rule, I feel it best to give two or three successive years' treatment before permitting omission of treatment.

DURING THE SEASON OR CURATIVE TREATMENT WITH POLLEN.

Frequently patients present themselves for treatment during their hay fever attack, and although pollen treatment at this time does not seem to be very logical, on the basis of anaphylaxis, the patient often will insist on taking the chance. Pollen treatment during the season does not seem logical because the patient is being injected with the pollen which is causing symptoms at the same time that he is being exposed to the pollen present in the air which he is inhaling. The danger resulting from large doses of the injected pollen is obvious because of an overdose, due to the combination of the injected pollen and the inhaled pollen. Therefore, in order that during the season treatment should be beneficial, the patient must be injected with minute amounts of the pollen extract in order to diminish artificially a few of the patient's antibodies, thus leaving a smaller number of antibodies in the patient for combination with the pollen antigen that is inhaled. If too much pollen extract (antigen) is injected, the patient should have symptoms due to over treatment alone, or he should be made worse, due to the injection of pollen extract (antigen) superimposed on the inhalation of pollen (antigen). It is evident that, on the basis of anaphylaxis, during the season treatment is hazardous, and although the skin test is the best guide as to the proper treatment, there is no way of controlling the amount of pollen that the patient may inhale.

The results from during the season treatment with grass pollen were: Of twenty-five patients treated, none were free from symptoms; seven, or 32 per cent, were practically free; four, or 18 per cent, were 75 per cent benefitted; three, or 13 per cent, were 50 per cent benefitted; the same number were 25 per cent benefitted, and five, or 22 per cent, were not benefitted. Since no patients were entirely relieved of symptoms, and since a large percentage were not benefitted, it is evident that preseasonal treatment insures better results by far than does during the season treatment. If preseasonal treatment is absolutely barred, during the season treatment is worth trying, provided it is given with sufficient care and the skin test is used as a guide to the initial treatment.

The results from during the season treatment with ragweed were: Of twenty-seven patients, three, or 11 per cent, were free from symptoms; one patient, or 3 per cent, was practically free; three patients, or 11 per cent, were 75 per cent benefitted; seven, or 25 per cent, were 50 per cent benefitted; one patient, or 3 per cent, was 25 per cent benefitted, and twelve patients, or 44 per cent, were not benefitted. Therefore, although a few patients were free from symptoms, and as many more were 75 per cent benefitted, the number who were only 50 per cent benefitted was as great as the number who were more than 50 per cent benefitted, and nearly half of the whole series were not benefitted at all. On comparing these results from during the season treatment with the results from preseasonal treatment, it is evident that for late hay fever by far the best results are obtained from preseasonal treatment, and it is questionable whether during the season treatment is even worth giving when it is taken into consideration that there are localities to which patients may go where ragweed does not exist.

PRECEDING AND DURING THE SEASON TREATMENT WITH POLLEN.

Some patients present themselves a few weeks previous to their season of symptoms, that is, they apply for treatment too late for preseasonal treatment alone and too early for during the season treatment. Rather than let them wait until their symptoms begin and then give them during the season treatment it is best to immediately begin treatment and continue the treatment on through their period of symptoms. This method of treatment yields better results than does the during the season treatment, but not as good results as the preseasonal treatment.

DURING THE SEASON TREATMENT WITH BACTERIA.

Occasionally when preseasonal pollen treatment fails, treatment during the season with autogenous nasal vaccine or a mixed streptococcus vaccine will benefit. The reason for such treatment is that it is quite possible that ragweed pollen exposure may in some cases cause such severe irritation of the mucous membranes that ever present bacteria may, either alone or together with ragweed pollen, be a cause of hay fever symptoms.

OTHER POLLENS THAT MAY CAUSE HAY FEVER.

It has been shown in this paper that the chief causes of hay fever occurring in the New England States were the pollens of ragweed, timothy and June grass; that an occasional cause is the pollen of the rose and redtop grass and of various trees. There is, however, almost an unlimited list of pollens that may at any time, but probably rarely, cause hay fever. Therefore, for those who treat a large number of hay fever patients and for those who fail to obtain satisfactory results from treatment with the common pollens mentioned, it is advisable to have a very extensive assortment of pollens. As a rule, however, it would seem to be sufficient to warn the ragweed patients that they should not smell the golden rod, golden glow, sunflower, poppy, aster, chrysanthemum and the like that pollinate during the ragweed season. The grass cases may be warned to avoid close contact with clover, lilies, daisy, dandelion, rose, lawn grass, orchard grass, corn and the like that pollinate during the grass season. In certain localities in this country it may be necessary to treat with the pollen extracts of the various grains, with sunflower, sage brush, or what not, but this necessity is not universal and is limited to the various localities where exposure to these particular pollens is unavoidable.

OTHER PARTS OF PLANTS THAT MAY CAUSE HAY FEVER.

The leaves of certain plants and trees may cause hay fever. The under surface of some leaves has a fine hairy growth, and these fine hairs may cause hay fever. I have observed one patient who had hay fever from and was sensitive to the hairs of the willow tree leaf; treatment with an extract of these leaves prevented hay fever. Another patient was sensitized to and had hay fever from the plantain lead. It is quite probable that the plane tree, the common shade tree in London, may cause hay fever. Such instances, however, are extremely rare and need not be considered, as a rule.

FOODS MAY CAUSE SEASONAL HAY FEVER.

Seasonal hay fever, due primarily to foods, has not come to my notice. However, during their season many hay fever patients find that certain foods aggravate their symptoms, whereas these same foods may be eaten without symptoms at a time when they are not accustomed to have hay fever, and they can eat the foods during their hay fever season, provided they have had sufficient pollen treatment to protect them against the particular pollen that primarily causes their hay fever. These foods are usually the fruits, and they have no apparent relationship to the causative pollen. Among the usual fruits should be mentioned peach, melon and apple. Green corn and the use of beer and wine often aggravate ragweed hay fever, but they do not aggravate grass pollen similarly. With the peach it is often the skin that causes trouble, whereas the pulp does not. The cooking of the fruits often renders them inert

ANIMAL EMANATIONS MAY CAUSE SEASONAL HAY FEVER.

The patient who is sensitive to animal emanations usually has symptoms on exposure to the animal at any and all times of the year; occasionally, however, such a patient has symptoms only from exposure to the animal during the spring and summer, when the animal is shedding, moulting or perspiring profusely.

BACTERIA MAY CAUSE SEASONAL HAY FEVER.

The possibility of bacterial infection as a complication of or secondary to pollen sensitization has already been discussed, and the possibility of bacterial infection as a primary cause of hay fever in patients who are not sensitive to pollens must be considered. Each year I examine three or four patients who have seasonal hay fever, but who are not sensitive to any of the pollens; therefore, these patients are treated with their autogenous nasal secretion vaccine.

PSEUDO HAY FEVER.

I have already, under perennial hay fever, discussed the possibility of pseudo hay fever which may complicate a seasonal hay fever as well.

CONCLUSIONS.

Satisfactory preseasonal pollen treatment yields excellent results in seasonal hay fever. By satisfactory treatment is meant five or six treatments with a 1:500 dilution of the pollen extract, or, better still, two or three treatments with a 1:100 dilution of the pollen extract.

When preseasonal pollen treatment fails, sometimes benefit results from during the season treatment with pollens and sometimes from during the season treatment with an autogenous nasal secretion vaccine. During the season treatment with pollens without preseasonal treatment is not very satisfactory, although such treatment for the early type of hay fever is worth doing provided, for some reason or other, it is not possible to give preseasonal treatment.

Treatment that of necessity must begin late preseasonally may be continued on through the pollen season with better results than those obtained by during the season treatment alone, but with much less beneficial results than those obtained by beginning preseasonal treatment early enough to permit of its discontinuance before the season of pollination begins.

Although in the New England States the pollen of ragweed (dwarf variety) is practically always the cause of hay fever (August and September), and the pollen of timothy grass is the cause of probably 90 per cent of the early hay fever (June and July), in other localities other pollens may play a great part in the cause of either type of hay fever. For this reason it is essential that other observers publish their tests, results and treatment in detail in order to learn the causative pollens and proper treatment, in various localities.

In the New England States the pollen of rose and redtop grass occasionally causes early hay fever, and treatment occasionally has to be given with these. The pollen of June grass is a more or less common cause of early hay fever, but when the season of its pollination is very early, unless the treatment is begun very early, too little treatment can be given with it to be of benefit, and when its pollination is very late sufficient treatment with timothy pollen has been given to protect against June grass pollen exposure.

Treatment with a combination of timothy and June grass pollens was not successful in my hands because of insufficient treatment with both pollens; the addition of June grass pollen retarded the amount of treatment that otherwise would have been given with timothy alone. Another illustration of the undesirability of mixed pollen treatment is noted with those patients who have both the early and the late types of hay fever. In these cases, during the month of May and part of June, large amounts of timothy pollen extract should be given together with small amounts of ragweed pollen extract, and the result in these cases is rather poor because there is a tendency to restrict the timothy treatment for fear of producing anaphylaxis from the combination of two pollens to which the patient is sensitive. In other words, treatment with a combination of pollens either diminishes the amount of treatment that is required with one or all of them, or pushing the treatment with the combinations leads to the danger of anaphylaxis For either or both of these reasons, I see no benefit to be derived from mixed pollen therapy. As regards the early type of hay fever, I consider it best to treat preseasonally with timothy or the chief causative pollen and, if necessary, to treat during the season with June grass pollen.

Since intimate exposure to other pollens which may be attributing causes of hay fever, the eating of fruits during the hay fever season, the possibility that bacteria play a part in the cause of hay fever and the exposure to olfactory irritants may all aggravate the symptoms of hay fever, it is remarkable that hay fever treatment is as near perfect as I have tried to present to you.

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XLI.

THE UNPLEASANT END-RESULTS IN WELL PERFORMED TONSILLECTOMIES.

By Charles W. Richardson, M. D., Sc. D.,

Washington.

The tendency of nature to replace tissues removed through accident or design; to restore function through complementary or compensatory action, are well recognized facts to all surgeons. This action is observable in connection with lymphatic tissue, especially when removed in early life. Especially is this to be noted in connection with the faucial and pharyngeal lymphatic tissues. Almost every one of my hearers have seen these facts illustrated in some of their operative cases or in the operative work of some of their equally skillful colleagues. It has seldom been the lot of any operator, however skillful he may be in his operative dexterity, to find all of his cases, one, two or three years thereafter, to have absolutely clear tonsillar fossæ, free entirely of evidences of lymphatic tissue. Even though the operative field may be clear, there is occasionally a case wherein there is an overgrowth of lymphatic tissue in close contiguity to the operative field. I do not wish to be placed in a position of stating that such occurrences are common, after thorough removal of the faucial tonsils, for such is not the case. I wish also to discriminate between those cases in which the tonsils have been thoroughly removed and those which have been imperfectly removed, which latter can be readily differentiated from the types to which I refer. It is a good practice to take advantage, which I always do, and examine every case that I have previously operated upon when the opportunity presents itself.

These few introductory remarks will bring us directly to the consideration of the unpleasant local end-results in well performed tonsillectomies.

The first group will include those efforts of nature to replace lymphatic tissue removed from the tonsillar fossæ by hypertrophy of glandular tissue in the immediate neighborhood.

A. Hypertrophy of glandular tissue in the lateral pharyngeal wall, posterior to the palatopharyngeal fold.

B. Hypertrophy of the lateral borders of the lingual tonsils.

C. Development or hypertrophy of glandular tissue in the palatopharyngeal fold.

In the first of the three groups above mentioned, I do not refer to simple showing of moderate hypertrophy of the follicle usually found in this area, but refer to those distinct tumorlike growths of lymphatic tissue which in their size and conformity resemble miniature tonsils.

In 1910 I saw the first evidence of this type of nature's attempt to replace a removed tissue in close proximity to the destroyed gland. This occurrence took place in a child of ten, upon whom I had several years before done apparently a perfect tonsillectomy. The case had been seen by a colleague, who promptly told the parents that I had not removed the left tonsil. They immediately called the attention of the fact to Dr. S. S. Adams, who referred the child back to me for reexamination. On the reexamination of the child I was surprised to note, in the left lateral wall immediately behind the posterior pillar, a large mass of lymphatic tissue perfectly resembling a faucial tonsil.

In June, 1914, I had a young girl of fourteen come to me on account of an acute inflammation of the left ear. On examining her fauces I found a perfect pair of tonsillar fossæ, but immediately behind the posterior pillar a similar growth of lymphatic tissue, though not quite as prominent as in the case just narrated.

On March 1, 1922, I had a young boy of twelve years referred to me by a distinguished member of this organization. In examining his fauces I found just behind the posterior pillar, in the lateral wall, a smaller but similar mass of hypertrophied lymphatic tissue.

The second group forms a most interesting series. I have seen six cases of this type, two in children and four in adults. The only one occurring following my own operative work was under my observation for aural treatment, twelve years after the removal of his tonsils. The patient stated that as it caused him no annoyance, he preferred not having it removed. In depressing the tongue, two dark reddish raspberry like masses were observed filling in the lower part of the tonsillar fossæ, giving the casual observer the impression that the lower lobe of the tonsil had not been removed. Depression of the tongue downward and forward caused the masses to move with the tongue, showing the fossæ free and demonstrating their true nature. The other three adults had discomfort and the masses were removed.

In the cases of the two children the condition was much more serious. The first case was that of a French child, male, twelve years of age, who was brought to me to have his tonsils reoperated upon. From his parents I learned that he had had what was thought to be a perfectly successful tonsillectomy done in Paris when he was eight years old. During the past year all of his old tonsillar symptoms had recurred. For several months he had had suffocating attacks at night, which were gradually increasing in their severity. Each night he would be aroused several times through attacks of "night terror." The child was anemic, under weight, and exceedingly nervous.

Examination of the fauces revealed large masses filling in the lower tonsillar fossæ. Depressing the tongue downward and forward caused these masses to follow the tongue and leave their position in the tonsillar fossæ. These masses were irregular, ragged nodular formations of a deep red color. Further investigation demonstrated that the tonsillar fossæ were free of tonsillar tissue and that the presenting masses were outgrowths from the lateral borders of the lingual tonsils. This adventitious tissue was removed. The masses were one and a half centimeter in diameter.

The second case was that of a female child, seven years of age, whose tonsils had been removed when she was four years of age. She had been examined by several physicians in Pittsburgh, all of whom believed that the tonsils had returned. The symptoms were similar but not as severe as in the case above narrated. The view obtained of the fauces and relation of the masses to the lingual tonsil were the same, excepting that the masses were not so large. They were removed.

Deposition of Lymphatic Tissue in Posterior Pillar.—I have seen three cases of large fusiform posterior pillars following perfectly performed tonsillectomies that had all the normal appearances of tonsillar tissue. Two of the cases I saw on account of acute follicular infection that had the characteristic appearance and all the symptoms that constitute acute follicular tonsillitis. In fact in one, my own case, during the attack I had to carefully examine the fauces before I could convince myself that the tonsillar fossa was clear.

In the healing process of enucleated tonsils, especially when the fossæ are very deep, there is a tendency to fill in the deep cavity by the development of granulation tissues. In most cases this granulating tissue is gradually absorbed in the nature process of repair, and we have a smooth, clean fossa. At other times this tissue undergoes organization, with the appearance here and there throughout its structure of lymphatic nodes. All of us have seen these cases, either in our own patients, or in the patients of our colleagues. There can be no doubt that granulation or true tonsillar tissue can develop in the tonsillar fossæ some months, even years, after a thoroughly well performed tonsillar operation. I have had under my observation on several occasions a woman for a recurrent subacute middle ear inflammation. She had had her tonsils removed in Chicago some five years before I first saw her, and I remember when I first viewed her fauces how I congratulated her upon the perfect result she had secured. I saw her several times at more than yearly intervals and always inspected those perfect tonsillar fossæ. Last spring she reported to me again for a few treatments, and at this time told me she had been having for several months some soreness in the left side of her throat. Imagine my surprise at noting in the lower portion of the left tonsillar fossæ a red inflamed mass nearly as large as the last phalanx ot my little finger.

Allied to the above mentioned group is the redevelopment of islands of tonsillar tissue to large masses of lymphatic tissue in the tonsillar fossæ. These small areas develop some period after the operation. It matters not how well the operation is done. All operators see these sequelæ. 1 see frequent evidence of this condition in very many cases of the best operators in the country. I have now a boy and a girl in their "teens," under my observation, who present this condition quite marked who were operated upon by a surgeon whom you and I recognize as the best operator in the United States. I had several years ago a case of this type that quite surprised me in several ways. The recurrence was no larger than a small pea, but displayed two follicles containing cheesy material. I had seen this child two years after a most perfect tonsillectomy, and all was perfect in the faucial fossæ. I never did a more perfect operation. Four years after the original operation I was confronted with the patient and the rather indignant mother, who stated that the family physician had said that I had never removed the left tonsil. The child lost weight, her color was bad, no appetite, and she had grown listless, tiring quickly. I examined the fauces and saw the small, pealike excrescence of lymphatic tissue in the bottom of the fossæ. While carrying on a running conversation I cocainized the excrescence and quickly pinched it off before they realized my intention. Within a few weeks the child returned to the normal. I mention this case to demonstrate that we must not always consider these cases too lightly.

Adhesion or shortening of the anterior pillar by its adherence to the dorsum of the tongue, and the adhesion of the lower lateral wall of the fossæ to the side of the tongue form another group. This group of accidents gives rise to very uncomfortable symptoms, if we are to accept the statements of patients. Extreme care is the watchword to prevent this accident. Occasionally it will occur in spite of our best endeavors.

Injuries of branches or the trunk of the hypoglossal nerve give rise to annoying symptoms. I have had one such case in my own practice and others in the practice of my colleagues.

My object in presenting this review of the unpleasant local end-results of well performed tonsillectomies is to make us more careful in our work along these lines so as to avoid as many of these pitfalls as possible, To make all endeavor to see our patients who have been operated upon six months or a year after the operation. To be more charitable to those with whom we work in a common field. Do not accuse any other man of having made an incomplete operation—no one can make such a statement with honesty unless he saw the operation performed.

XLII.

A CONTRIBUTION TO THE STUDY OF BRAIN ABSCESS.

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In reviewing the subject of brain abscess one is struck by the scarcity of symptoms observed, and the following paper represents an attempt to ascertain their relative frequency and diagnostic value. Most of the case histories I have been able to study are somewhat meager, and in many cases one cannot assume that the lack of specific mention of certain symptoms means that they were absent. They were probably not observed. Yet, even in the case where negative findings are definitely stated there is usually relatively little upon which to base a definite diagnosis. The clinical picture of brain abscess presents a wide variety of symptoms and signs, but this picture is a composite one, and one can never expect to find more than a few symptoms in any one case and they are often fleeting or nebulous. Symptoms of increased intracranial pressure are, as a rule, fairly definite. These, however, may be due to extradural abscess, cerebral abscess, cerebellar abscess, tumor or meningitis. The more acute the condition the more violent are the symptoms. Thus, in tumor, which generally takes months to grow, although the mass may be large, signs of increased pressure may not be so marked as they are in the case of abscess, or still more in the case of meningities, where the evolution may occur within a few days.

In this paper I propose to give an analysis of the histories of 67 cases of otogenic brain abscess, obtained from the literature. Detailed notes of a recent case of cerebellar encephalitis and one of temporosphenoidal abscess are also added.

FREQUENCY OF OCCURRENCE.

Taking the figures supplied by the Royal Infirmary of Edinburgh reports for the five years 1914-18 inclusive[1] we find that out of a total of 3,918 cases of middle ear suppuration, including dried up cases but not including acute or chronic catarrh, there were 85 (2.19 per cent) cases with intracranial complications. Of these 27 (0.58 per cent) occurred in acute cases and 58 (1.4 per cent) in chronic cases. Out of these 85 cases 15 were brain abscess. The frequency then of the occurrence of brain abscess in relation to otitis media is 15 to 3,918, or 0.38 per cent, that is, about 4 per thousand. It will be noted incidentally that intracranial complications occur more than twice as often in chronic otitis media as in acute. Logan Turner in 1909[2] mentions 14 intracranial complications in 532 cases of middle ear suppuration (2.6 per cent). Out of these three were brain abscesses (0.5 per cent). Mygind[3] in 207 cases of otogenic intracranial disease found meningitis 141 times, sinus thrombosis 106 times, brain abscess 42 times and subdural abscess 19 times. Multiple complications were twice as common in chronic cases as in acute (48 to 25).

TRACKS OF INFECTION FROM MIDDLE EAR.

In the case of temporosphenoidal abscess, probably by far the greater number are caused by direct spread of infection upwards through the tegmen tympani. In most of the recorded cases which were available for study the tegmen was found to be eroded and the dura covered with granulations. Here a localized patch of meningitis is set up, the pia arachnoid becomes adherent to the dura and the process spreads into the brain itself, forming a subdural abscess. Vessels on the surface of the brain become thrombosed and cause septic softening in the white matter. In other cases, however, the tegmen is not eroded, and when the dura is exposed it appears healthy except for increase of tension. The brain is not by any means always adherent to the membranes when there is an abscess underneath. The surface frequently looks quite normal. In such cases it is more than likely that the abscess originates in a small septic embolus. That part of the temporosphenoidal lobe which lies over the tegmen represents one of the boundary zones in the cerebral circulation, where the regions supplied by the middle and inferior cerebral arteries meet. (Fig. I.) The vessels here are hence all of small caliber and easily permeable. The veins of this region require special study.

The great bulk of the venous blood from the brain leaves the skull by the sigmoid sinus and internal jugular vein of each side. However, there are numerous accessory outlets which act as safety overflows. These are the mastoid foramen with its emissary vein and the posterior condylar foramen, both from the lateral or sigmoid sinus. The inferior petrosal sinus leaves the skull through a separate compartment of the jugular fossa.

There is also a free communication between the cavernous sinus and the pterygoid plexus on each side through the foramen ovale. The superior petrosal sinus, which is relatively small, forms a communication between the cavernous sinus and the genu of the lateral sinus on each side. The blood flows away from the cavernous sinus towards the sigmoid. However, owing to its oblique entrance into the latter, its flow must necessarily be retarded, and under varying conditions of position and pressure it is probable that the flow is occasionally reversed. The superior petrosal receives several veins from the lower surface of the temporosphenoidal lobe and also a small but constant group of vessels which collect from the mucous membrane of the aditus and antrum and pass through the arch formed by the superior semicircular canal to emerge from the bone in the subarcuate fossa on the posterior surface of the petrous pyramid. In the infant this fossa is as big as the internal auditory meatus and transmits a large vein. This gradually lessens in size, till in the adult it is barely visible. The superior and inferior petrosal sinuses also receive some large cerebellar veins, which come from between the superior semilunar lobule and the posterior part of the quadrangular lobule.

In the course of a middle ear suppuration pathogenic organisms may pass along the subarcuate vessels into the superior petrosal sinus and thence into the thin walled cerebral and cerebellar tributaries. From here they may pass into the subarachnoid space or even into the brain substance itself. A case has been described by Fraser,[4] in which infection passing along the subarcuate vessels was demonstrated microscopically.

It is interesting to note from the records available that out of 67 cases of brain abscess, of which 43 were in the temporosphenoidal lobe and 24 in the cerebellum, sinus thrombosis was associated in 16 and perisinus abscess in 4. In the 43 temporosphenoidal abscess cases thrombosis was present in only four and perisinus abscess in only one. It is to be noted also that in one of these the brain abscess was on the opposite side from the ear lesion. In the 24 cerebellar cases thrombosis was associated in 12 instances, while in three others there was a perisinus abscess without thrombosis. This rather points to the probability of venous embolism as an etiologic factor in the formation of abscess, in the cerebellum at any rate.

FREQUENCY OF COMPLICATIONS.

According to Milligan,[5] in acute cases extradural abscess or sinus thrombosis is the route by which infection reaches the cerebellum. In chronic cases the abscess is generally secondary to labyrinthitis.

As far as one may generalize from the numbers before me, temporosphenoidal abscess is less frequently accompanied by other complications than cerebellar abscess. There were seventeen cases of temporosphenoidal abscess without other complications. There were five in which more than one abscess was present, and included in these five are two where cerebellar abscess was present in addition. Eleven cases had some other complication, such as extradural abscess, sinus thrombosis, or meningitis Cerebellar abscess unaccompanied by other conditions was found in only three cases, multiple abscess in three cases, including the two already mentioned with temporosphenoidal abscess in addition. Cerebellar abscess and labyrinthitis occurred together three times, and cerebellar abscess with extradural abscess, sinus thrombosis or meningitis in eight cases. There was one case of subdural cerebellar abscess with extradural abscess.

From the foregoing it will be seen that in roughly half of the temporosphenoidal cases the abscess occurred without other complications. In cerebellar abscess, on the contrary, only three out of seventeen were found without other complications.

PATHOLOGY.

When I was in France I had the opportunity of carrying out some 72 postmortem examinations on head cases. With the exception of a few cases of cerebrospinal meningitis, these were all gunshot wounds of the brain. The brain lesions seen bore a great resemblance to the otogenic brain abscesses of civil life. In almost every case there was some attempt at the formation of adhesions at the edge of the opening in the dura. In the most recent cases there was of course a great deal of damage to the brain for some distance round the actual track of the projectile. In cases which had lived for several days or longer a great deal of the damaged brain tissue had sloughed out, leaving an abscess cavity. This at first was irregular in outline, but older cases showed the formation of a fairly well marked capsule with smooth lining walls, showing a great tendency for the limitation and ultimate shutting off of the damaged area.

In the case of otogenic brain abscess the first stage is a localized patch of red softening or encephalitis. This later breaks down and forms an abscess cavity. In many cases there is a distinct tendency for the limitation of the spread of the infection and a more or less encapsulated abscess results. In other cases the encephalitis extends rapidly and causes death from increase in the intracranial pressure. An encysted abscess which had been more or less latent may suddenly become surrounded by a zone of acute encephalitis with subsequent secondary abscess formation, as is shown by Macewen's case XXX, in which the encapsulated abscess was found inside a more recent surrounding abscess.[6]

INJURY AS AN ETIOLOGIC FACTOR.

Five of the cases under review had received some injury to the head sufficient to cause loss of consciousness. This appeared to play some part in the formation of the abscess, as the symptoms dated from the injury in most of them. It is only reasonable to suppose that when an injury occurs during the course of a middle ear suppuration new avenues of infection may be opened up.

With regard to the question of trauma, the mastoid operation must also be considered. In seven cases a mastoid operation had been done with apparently no suspicion of the presence of a brain abscess. After a period varying from some hours to a month symptoms of brain abscess began to show themselves. If one asks why, he is faced with three alternatives: did the trauma of the mastoid operation cause the brain abscess, or did it light up an already existing latent abscess, or had it nothing to do with it? These questions are difficult if not impossible to settle from the small number of cases available for study. On the other hand, in several instances patients were seen with symptoms leading to suspicion of an intracranial complication. These disappeared entirely after operation for one or two weeks, only to reappear along with other more definite signs. The question then must be left open for the present. The latent period after mastoid operation requires investigation, as it is obvious from numerous cases that the abscess was already present at the time of operation, yet all the suspicious symptoms disappeared for a while, leading the surgeon to think he had been mistaken.

CAUSE OF DEATH.

In a study of 25 fatal cases of brain abscess in the available literature it was seen that diffuse purulent meningitis was the cause of death in twelve. In two of these the meningitis had been set up by leakage from the abscess into the subarachnoid space. Five cases died from pressure symptoms, in three of which death was due to acute encephalitis or edema. In three cases the patients died with pressure symptoms due to a second undiscovered abscess. In three death was due to rupture of the abscess into one of the ventricles. In my own experience this latter complication is generally fatal within a few hours, but in one of Fraser's cases death did not occur till 17 days after rupture.

GENERAL SYMPTOMS OF BRAIN ABSCESS.

It must be clearly understood that there is no one symptom or sign which is constantly present. By analyzing a large series of cases a fairly definite composite picture may be formed, but it is unusual to get more than a very small proportion of the symptoms and signs in any one case.

Brain abscess is generally described as showing three clinical phases: (1) An initial, (2) a middle, and (3) a terminal stage. The first stage is very seldom seen in hospital. It corresponds with the formation of an area of red softening and is generally ushered in by a chill, headache and vomiting. The chill may be merely a feeling of cold with cutis anserina, or may be an actual rigor. The temperature may be slightly raised. In the middle stage the signs of increased pressure predominate. The senses are blunted, the cerebration is slow and the mind is dull. Headache is severe but does not cause outcry. The pulse and respiration rates are slow, and the temperature is normal or subnormal. The terminal stage is short and is characterized by coma, incontinence and increasing respiratory failure. Hyperpyrexia and rapid pulse may immediately precede death.

Encephalitis, abscess and meningitis give a series of symptoms which are common to each, but varying in degree. The symptoms are in the main due to the accompanying increased intracranial pressure. These are headache, vomiting, mental dullness, drowsiness, optic neuritis, slow pulse and often subnormal temperature. In many of Macewen's cases the onset of symptoms was ushered in by a rigor or feeling of cold (ten out of nineteen). However, in four of them there was sinus thrombosis, and in several others there was a perisinus abscess with a suspicion of involvement of the sinus. In the forty-seven other cases here analyzed rigor or chilly feeling was only mentioned five times, and in three of these there was sinus thrombosis. That is, out of sixty-six cases in all, rigors occurred in fifteen, and of these fifteen, nine were cases with sinus thrombosis. From this it would appear that rigors or chills sometimes occur with simple brain abscess, but that in most cases where such a symptom is noted it is due to an accompanying sinus thrombosis.

Headache is one of the most constant symptoms and was present in fifty cases. In some of the remaining cases its presence or absence could not be ascertained, as the patient was already comatose. Where details were given the headache had usually commenced on the affected side and was extremely acute. In the initial stage it was noted in several cases as being so severe as to prevent sleep. Later when the patient was passing into the second stage it was generally frontal or occipital, or both. The headache of abscess is extremely severe, with exacerbations at times. It does not, however, as a rule cause the patient to cry out as it does in meningitis, where the headache is excruciating. This is so, even with the socalled serous meningitis. Aboulker[7] emphasizes the difference between the headache of abscess and that of meningitis. The patient with abscess, when interrogated, will tell you that he has a terrible headache, but he does not, as a rule, volunteer the information.

Mental disturbances manifested in several ways have been described by various observers, as slowness of cerebration, obnubilation or clouding of the intellect, apparent stupidity, absent-mindedness, lack of attention, psychic depression, etc. In several instances noted the mental condition was practically the only symptom of the abscess. In one case reported by Aboulker a girl, aged 15, had been attending a clinic with chronic middle ear suppuration. It was noticed that she was easily fatigued by questions and was very absent minded. On the strength of this, cerebral abscess was diagnosed, although there was no headache or any other sign, and lumbar puncture was normal. The patient, however, refused to go into the hospital, but returned a month later with mastoiditis. An abscess was found in the temporosphenoidal lobe and another one in the cerebellum. Voisin[8] reports the case of a soldier who was thought by his comrades to be feeble minded. He was admitted to the hospital with acute middle ear suppuration, and he also had an abscess in the right tonsil, with some delirium. Some days later there was a little transitory mental confusion; ten days later he suddenly developed torpor and hemiplegia and died in 48 hours. At the postmortem an abscess the size of an egg was found in the left ascending parietal region. Another was forming in the occipital lobe. He had never complained of headache or any other sign. Adson[9] notes symptoms of mental disturbance in fifteen of his twenty-six cases. They were lack of attention, loss of memory, delirium and coma. This clouding of the intellect, inability for sustained attention, and difficulty in exerting one's strength are due to the increased intracranial pressure, and also to the presence of a large amount of toxin in the cerebrospinal fluid. As a rule these are not noticed till the second stage, when signs of drowsiness predominate.

Drowsiness is not found in the early stages, as a rule, but only in the second and final stages. As the intracranial pressure increases, so does somnolence till it merges into coma. In the second stage the patient spends most of his time lying half asleep. He can be roused and will answer questions, but immediately drops off to sleep again.

Vomiting occurs in the early and middle stages, but is rarer in the last stage. In the cases under review it was noted as a symptom thirty times. However, it was doubtless present in many more of the cases, as in those which were carefully detailed it was always there. The characteristic of vomiting due to brain abscess is that it bears very little relation to the taking of food. As far as the act of vomiting itself is concerned, it is like that from any other cause, but is usually unaccompanied by nausea. Projectile vomiting, socalled, is not very common. It was noted as such in only one case. The vomiting in the initial stages of brain abscess and in sinus thrombosis and meningitis frequently accompanies a rigor. Moving the patient in bed during an examination very often brings it about.

Nausea, which is so marked a feature of labyrinthitis, is generally absent in cerebral abscess, though sometimes present in cerebellar abscess.

Obstinate constipation is a frequent symptom when the intracranial pressure becomes great. The tongue is also often very thickly furred.

Optic Neuritis.—Adson (loc. cit.) found choked discs in nine out of his twenty-six cases. Logan Turner (loc. cit.), in fourteen cases of intracranial complications of otitis media, found optic neuritis in none of them, though in some there was blurring of the edges of the discs and engorgement of the vessels. The condition of the eye grounds was noted in thirty-four cases in this series. Double optic neuritis occurred in twelve, and in three of these it was more marked on the affected side, while in one it was more marked on the opposite side. In seven cases there was optic neuritis on one side only. In six of these it was on the affected side, and in one on the opposite side. There was blurring of the discs in four cases, and engorgement of the vessels in four cases, in one of which it was more marked on the opposite side. The discs were normal in eight cases. That is to say, in twenty-six of the thirty-four cases there was a positive finding, and the condition was generally more marked on the affected side. Optic neuritis is not, as a rule, seen in cases where the increase of intracranial pressure had lasted less than a fortnight. In two of the cases under review there was no neuritis when first examined, but it developed some time later. In a case which I saw some years ago, after repeated examinations neuritis was seen to develop only two weeks after the onset of symptoms. Optic neuritis appears earlier and is more severe in posterior fossa infections than in those of the middle or anterior fossa. Blindness is also said to be much commoner as a result of cerebellar than of temporosphenoidal abscess. According to Macewen, the optic neuritis is rarely followed by atrophy, and the sight generally returns when the brain condition is relieved. From the foregoing it will be seen that the absence of optic neuritis does not rule out the diagnosis of brain abscess, but its presence is an important confirmatory sign.

Pupils.—In the early irritative stages the pupils are contracted and active. When pressure symptoms begin to predominate they tend to dilate and are sluggish in their reaction to light. The state of the pupils was noted in twenty-one cases. In five of these, both pupils were dilated and fixed, and in all of them other pressure symptoms were very marked. In six they were equal and small, and in all of these except one pressure signs were not severe. The mental state was rather irritable than dull. In the one case, a fatal one with double cerebellar abscess, the pupils became contracted just before death. In ten instances the pupil on the affected side was dilated and in most of them it was fixed. In one of the cases the pupil was active and the patient was restless. In the others the patients were dull and showing signs of severe pressure. Macewen states that when the abscess is in the frontal or temporosphenoidal lobe the pupils may be either small or large, but especially sluggish. Sometimes the only difference is that the pupil on the affected side is more sluggish. In diffuse meningitis the pupils are usually equal and contracted till a late stage, when they dilate.

Pulse.—In the initial stage of brain abscess the pulse may be frequent, but in the stage when cases are usually seen the pulse is generally slow. A pulse rate of 50 to 70 is very common. In this series the condition of the pulse was noted in forty cases. In four of them the lowest readings were between 40 and 49, in ten between 50 and 59, in eleven between 60 and 69, in none between 70 and 79, in two between 80 and 89, in four between 90 and 100 and over, one was noted as being normal and six were noted as subnormal. In the group over 90, one was a child of 8, who died. The other three were uncomplicated cases in adults who recovered. The four cases with a pulse rate between 40 and 49 were also uncomplicated. From the above figures it would appear that the tendency is to remain subnormal and that accompanying complications or intercurrent disease do not have any marked effect on this tendency. When an abscess ruptures, either on to the surface of the brain or into the ventricles, there is generally a marked acceleration before death, as is shown by the case of Fraser's (loc. cit.), where the pulse rose to 120 and temperature to 104, and another in which the pulse was 130 and temperature 104, and in Macewen's case XXX, where the pulse was 140 and temperature 105.8.

Temperature.—It is more difficult to generalize on the question of temperature, as it is much more variable than the pulse, but in a large number of uncomplicated cases it tends to be subnormal. When other pressure signs are present, 97° F. is common. However, when meningitis exists as well, the temperature may remain above normal, and the signs of abscess may be completely masked. Meningitis is characterized by a high temperature, with a normal or subnormal pulse rate. As already noted in connection with the pulse, after rupture of an abscess into the ventricle, hyperpyrexia is the rule. I have seen a case with temperature of 107 just before death.

Respiration.—The respiration rate is generally about normal with a tendency to slowing. When the intracranial pressure is becoming severe and the patient is becoming comatose, Cheyne-Stokes respiration or other intermittent breathing is common. Intermissions between breaths may last half a minute. When death from increased pressure is approaching, the respiratory function is usually the first to go. Patients can be kept alive for hours if artificial respiration is carried out. During operation there is very frequently cessation of breathing, necessitating artificial respiration till the abscess is opened. This is particularly so in cerebellar cases.

Leucocyte Count.—Darling has studied the blood picture in a large series of ear cases and the results of his investigations are quoted here.[10] In acute middle ear suppuration with mastoiditis the average white blood count was 12,400 per cub. mm.; maximum, 20,600; minimum, 7,200. In chronic middle ear suppuration the average was 10,200. In chronic middle ear suppuration with mastoiditis the average was 17,600; maximum, 22,100; minimum, 12,700. General anesthesia, particularly with chloroform and ether, causes an increase in the leucocytes of about 8,000 for a period averaging three and one-half days. In his series there were seventeen intracranial cases. In extradural abscess the average was 18,400; maximum, 30,000; minimum, 9,300. In six brain abscesses the average was 12,500; maximum, 14,000; minimum, 10,300. In meningitis the average was 25,500; maximum, 33,000; minimum, 17,000. Sinus thrombosis averaged 14,500, but, leaving out one atypical case, it was 17,000. Darling states that if the polymorphs are under 77 per cent, probably no intracranial complication is present. Single examinations are not so useful as repeated ones, as it is only then that the change in the blood picture gives valuable information. Adson gives an average of 16,000 in brain abscess cases. In some instances the count was normal.

From the foregoing it is evident that the leucocyte count does not help materially in the diagnosis of brain abscess. In obscure cases, in which one is uncertain as to the presence or absence of a latent abscess, the mastoid condition alone would give a blood picture very similar to that of brain abscess. However, as one can exclude extradural abscess and meningitis by other means, and if the uncertainty occurs more than five days after operation or in the absence of acute mastoiditis, a leucocyte count of 12,000 to 16,000 would suggest the presence of abscess.

Cerebrospinal Fluid.—Adson states that when there is no diffuse encephalitis or meningitis the cell count in the cerebrospinal fluid is normal in brain abscess (2 to 5 lymphocytes per cub. mm.). In cases complicated with meningitis it may be as high as 108,000. Fleischmann[11] in an extensive series of examinations of the cerebrospinal fluid in ear cases had twelve of brain abscess, of which seven were otogenic. There were twenty-three lumbar punctures in all. He found the pressure increased in most of the cases, though not in all. There was no increase of the cells in three, but in ten there was a marked increase of lymphocytes up to between 30 and 300. The total albumin and globulin was increased, though not constantly nor to a marked degree, the normal albumin content being about 0.2 to 0.5 per cent. Sugar is present in brain abscess cases, but is generally absent in meningitis. The change in the number of cells is the most constant sign.

Lumbar puncture had been performed in twenty-four of the cases in this series one or more times. The results were so variable and the points noted and omitted so different that one cannot draw any very definite conclusions, except that the pressure was in most cases raised. In those cases where the cell count was mentioned it was increased, and in quite a number the increase was enough to cause turbidity, yet no organisms could be seen on direct smears, and cultures remained sterile. In what the French writers describe as méningite puriforme aseptique a similar condition is found, and Aboulker (loc. cit.) states that in it the symptoms are much more alarming than in the much more serious brain abscess. The headache is much more intense, etc.

In other cases the fluid was clear, but contained cocci of various sorts. While the presence of germs in the cerebrospinal fluid must always be regarded as an ominous sign, these cases are by no means always fatal, as there were four in this series alone which recovered.

In a case of temporosphenoidal abscess reported by Fournioux,[12] in which meningitic symptoms were present, the first lumbar puncture showed nothing abnormal. After a radical mastoid operation the symptoms subsided. Nine days later there were again meningitic symptoms and a brain abscess was opened. Next day the cerebrospinal fluid was turbid, but the pressure was not increased. There was a heavy deposit with 80 per cent polymorphs, 15 per cent lymphocytes, and 5 per cent endothelial cells. Albumin was present; no glucose; some cocci but culture negative. Four days later cerebrospinal fluid unchanged. Eight days later, flocculent deposit; polymorps 70 per cent; albumin increased; sugar absent; germs present.

Twelve days later a trace of sugar present; lymphocytes 90 per cent. Recovery uneventful.

So much for the general symptoms and signs of brain abscess. Let us now consider the symptoms and signs which help us to determine the site of the abscess. They comprise to a great extent irritative and paralytic phenomena, but also disturbances of speech and other mental functions in the case of the cerebrum, and troubles in coordination, balance, etc., in the case of the cerebellum.

Localized Pain.—Macewen draws attention to the value of percussion of the head in locating the site of an abscess. Patients with brain abscess generally resent tapping over the region of the abscess, while they do not mind it over the rest of the skull. Adson states that he found local tenderness in twelve of his twenty-six cases; in four of these it was not over the site of the abscess, but in the remaining eight it was a confirmatory sign. In several of the other cases reviewed here tenderness to percussion was noted.

TEMPOROSPHENOIDAL ABSCESS.

In addition to the general symptoms mentioned above, temporosphenoidal abscess often shows some localizing signs, such as paralysis on the opposite side, convulsions, aphasia, etc.

Paralysis.—In forty-three cases of temporosphenoidal abscess hemiplegia of the oposite side was noted in seven, and weakness of the opposite side in three. The oculomotor nerve of the same side was involved in five cases. In four of these there was ptosis, and in the other case the whole of the nerve was paralyzed except for the branch to the levator palpebræ. In one case the sixth nerve of the same side, and in one that of the opposite side was paralyzed.

Paralyses are less common in cerebellar abscesses, but when paralysis or paresis is present it is on the same side as the abscess, in contradistinction to temporosphenoidal abscess. Weakness of the arm of the same side was noted in three cases only. In one of these the paralysis of the limb was complete, but function was restored after the abscess was opened. There was paralysis of the sixth nerve on the same side in two cerebellar cases and of the oculomotor in one. From this it might appear that temporosphenoidal abscess shows more localizing signs than cerebellar abscess. This is, however, not the case, as the cerebellar localizing signs are of a different category and will be discussed later.

The paralysis or paresis of the limbs of the opposite sides in temporosphenoidal abscess hardly require comment, except that if present it is important to note the order of involvement of the limbs. If face, arm, leg is the order of involvement the chances are that we are dealing with a subdural abscess or meningitis. If in the opposite order it is more likely to be pressure on the internal capsule. If the infective process extends as far as the basal ganglia, part of the lenticular or thalamic syndrome of Déjerine and Roussy may be met with. This syndrome consists of hemiataxia with marked disturbances of deep sensation with slight hemiparesis. Hemichorea and hemiathetosis may be associated. Macewen's case XXIX showed paralysis of the arm and leg of the opposite side, accompanied by choreic movements. Paralysis of the third or sixth nerve of the same side may be due to pressure on the nerve trunks or involvement of the peduncle or basal meningitis.

Paralysis of the cranial nerves is more often due to basal meningitis than to brain abscess. Paralysis of the sixth nerve on the affected side occurs not infrequently as a result of localized pachymeningitis at the apex of the petrous bone with headache and a few other symptoms, and this is known as the syndrome of Gradenigo. It is not usually fatal but only transitory.

Reflexes.—The examination of the reflexes as a rule gives very little help in the diagnosis of brain abscess. In temporosphenoidal abscess the knee jerks are sometimes altered on the opposite side, but generally only when there are already paralytic phenomena. In cerebellar abscess the knee jerks are usually unaltered.

Convulsions, etc.—Adson reports Jacksonian epileptic attacks in twelve and grand mal epilepsy in four of his twenty-six cases. In the series studied here, convulsive movements of the limbs were noted in eight of the sixty-seven cases. Three of these had repeated generalized convulsions, and in two of the three the patients had meningitis as well. Jacksonian fits or twitching of the limbs on the opposite side occurred in three cases. Seven of the eight cases were temporosphenoidal abscess. The remaining case was one of cerebellar abscess complicated by basal meningitis. Apparently, then, convulsions or jerkings are not very common in uncomplicated brain abscess, but when present are of considerable value in localizing the condition. Twitchings are rare in cerebellar abscess.

Disturbances of Speech.—The modern conceptions of aphasia advanced by Pierre Marie have been fully substantiated, according to Chatelin,[13] by the study of gunshot wounds of the brain occurring during the great war. Briefly, Marie distinguishes between aphasia proper and motor aphasia, which he terms anarthria or dysarthria, according to the degree of the disability. In the latter condition the comprehension of speech is retained. Aphasia is the loss of understanding of speech, and may consist in a difficulty in finding words to name objects or convey ideas, using wrong words (paraphasia) or, on the other hand, there may be a lack of comprehension of words heard. In alexia, or visual aphasia, or psychic blindness, there is a lack of comprehension of things seen. There is always more or less clouding of the intellect associated with these different forms.

Anarthria is associated with the lenticular zone of Pierre Marie, which is defined as including the central gray nuclei, the internal and external capsules, the insula, the motor cortex and some of the important white fibers. The third frontal convolution or area of Broca is not included. Anarthria is not connected entirely with the left side of the brain, but is also found in corresponding lesions of the right side.

True aphasia is associated exclusively with the left side of the brain in right handed individuals. It is located in the area of Wernicke, which includes the gyrus supramarginalis, gyrus angularis, the posterior end of the two first temporal convolutions and the subjacent white matter as far as the wall of the descending horn of the lateral ventricle, which immediately adjoins the already mentioned lenticular zone.

Lesions in the motor area, namely, ascending frontal convolution, produce anarthria with or without accompanying paralyses. Lesions of the left temporoparietal region in the area of the supramarginal gyrus produce aphasia with some anarthria. Lesions in the middle and posterior parts of the temporal convolutions produce a difficulty in naming objects. The intellectual faculties are very much affected, especially that of calculation. There is very little anarthria. A lesion in the left posterior temporal and angular convolutions produces alexia predominantly, frequently associated with hemianopsia if the lesion is deep. Dysarthria or difficulty of speech may also be caused by cerebellar or bulbar lesions.

In thirty-one temporosphenoidal abscesses there were fifteen on the right side, thirteen on the left side, and in three instances the side was not stated. Aphasia was noted in seven of the thirteen. Macewen's patient XX, who had an extensive necrosis of the left temporosphenoidal lobe, could not understand speech properly and mistook the meaning. There was paraphasia, or use of the wrong words, and considerable mental obscuration as well. In another case (Fraser) a child could name some objects but not others. She could name knife but could not name keys, though she said they were for opening doors. In another case (Macewen, XXXVII), where there was a cerebellar abscess and at the same time a large subdural abscess over the first and second temporal convolutions, there was complete word deafness. The patient only arrived at the meaning by repeated guessing, but he had complete command of language to answer. Later, the same patient developed visual aphasia, or psychic blindness. He could not recognize objects held up to him till he touched them. In one case of cerebellar abscess "motor aphasia" was noted. This was probably dysarthria connected with ataxia and other disturbances of coordination.

CEREBELLAR ABSCESS.

Cerebellar abscess is much richer in localizing symptoms and signs than temporosphenoidal abscess. Among the symptoms and signs are nystagmus, vertigo, troubles of balancing and coordination, such as falling, pointing error, ataxia, lack of muscular tone on the affected side, intention tremor, etc. Repeated yawning and rigidity of the masseters were frequently observed by Macewen.

Nystagmus, apart from that of ocular origin, which is easily distinguished, is almost invariably due to a lesion of an irritative or destructive character, either in the labyrinth or in the posterior cranial fossa. The commonest cause of labyrinthine irritation is an erosion of the labyrinth wall, generally in the region of the external semicircular canal. The labyrinth is functionally intact and the condition is easily diagnosed. The nystagmus and giddiness are only occasional and are generally produced by sudden movements of the head. The detailed examination need not be discussed here. Destructive lesions of one labyrinth cause violent dizziness and nystagmus, accompanied by nausea and vomiting. The nystagmus of destructive lesions of the labyrinth is invariably directed away from the affected side—that is, the slow or labyrinthine movement is towards the affected side, and the quick or cerebral movement towards the opposite side. The nystagmus is mixed in character—i. e., rotatory and horizontal. Labyrinthine nystagmus is most marked at first, but gradually diminishes from day to day and finally disappears. The labyrinth does not respond to stimulation by the caloric or rotation tests and is absolutely deaf. Associated with nystagmus to the opposite side is a tendency to fall to the diseased side, or to deviate towards the diseased side in walking. Romberg's test gives falling to the diseased side. If the head is turned away from the diseased side the patient falls forwards; if turned towards the diseased side he falls backwards. Barany's pointing test gives deviation towards the diseased side with one or both arms.

Cerebellar nystagmus differs in several important respects from labyrinthine nystagmus. In the first place, it is as a rule not continuous. One day it may be present and another day absent. Its direction also may vary from time to time; one day it may be directed to the right and another day to the left. In a case of Fraser's[14] there was spontaneous nystagmus one day to the sound side of the first degree; next day there was nystagmus of the second degree to the affected side. Nystagmus due to cerebellar lesions does not rapidly disappear as does labyrinthine, but on the contrary lasts a long time and tends to increase. In cerebellar lesions there is a tendency to fall or deviate to the affected side. This falling tendency is uninfluenced by altering the position of the head, and according to Holmes[15] is unaffected by closing the eyes. In cerebellar lesions the pointing test generally shows a deviation of the arm on the affected side, and the direction of the deviation has no relation to the direction of the nystagmus if the latter is present. The deviation is generally towards the affected side. Gordon Holmes in his observations on war wounds of the cerebellum has done much to systematize the symptoms and signs. He states that nystagmus is almost invariably present and points out a further characteristic of cerebellar nystagmus. "It is essentially a fixation nystagmus—that is, it can be seen as a rule only when the patient fixes an object." There is a rest point towards which the eyes tend to deviate when the patient is at rest. This point is usually 10 to 30 degrees to the unaffected side of the middle line. When the patient is asked to look at an object straight in front of him, the eyes tend to roll slowly to the rest point on the unaffected side, and are brought back by quick jerks. On looking towards the affected side the nystagmus becomes much more marked.

Labyrinthine nystagmus, on the other hand, is best seen when the patient does not fix an object. On fixation the nystagmus may be controlled and may disappear.

Loss of muscular tone is a constant feature of cerebellar lesions. This is evidenced by the unnatural attitudes assumed by the limbs on the affected side. If the limbs are passively moved the affected ones flop about much more than those of the other side. The knee jerks are unchanged.

There is delay in initiating and stopping any voluntary movement. This is brought out by asking the patient to perform rapidly alternating movements with both hands simultaneously. The limb on the affected side lags behind (adiadochokinesis).

Disturbances of coordination when analyzed are resolved into (1) asynergia, or defective synchronization of the muscles, resulting in intention tremor; (2) dysmetria, or failure to gauge the force and extent of a desired movement.

There are many other signs deserving mention, but time and space do not allow of it at present. For the same reason the significance and interpretation of the vestibular reactions cannot be discussed, except to state that the condition of the labyrinth must in all suspicious cases be thoroughly investigated. As already mentioned, cerebellar abscess is very frequently associated with labyrinthitis, and merely to discover that one labyrinth is not functioning is not enough . The vestibular reactions must be analyzed to ascertain whether there is any cerebellar lesion as well.

REPORT OF A CASE OF CEREBELLAR ENCEPHALITIS.

A boy, aged 17, was seen on April 15, 1921, with the following history. His left ear had been running all his life. He had had attention for this from time to time, and the condition had improved. At Easter he had had a knockout blow while boxing and had not been quite well since then. Five days ago he had been out in a cold wind and soon afterwards began to have some pain in the left ear, which soon became very acute, and the discharge became very profuse. He remained several days in the school sickroom and came home on the 14th. On the journey home in the train he suffered from an extremely severe headache on the top of his head, with great dizziness and vomiting. Since arriving home had vomited several times and had some chilly feelings. The headache was still present on the top of the head and had been constant for two days.

On examination there was copious purulent discharge from the left ear with distinct mastoid tenderness. The whole left drum membrane was gone; right drum membrane normal; very deaf in left ear; could hear raised voice with noisebox in right ear; temperature 101.5 F. It was higher the night before. Nystagmus absent; pointing test normal; no adiadochokinesis.

Operation: Cortex thin; bone extensively softened and extremely vascular; cells not numerous, but some pus in them; antrum also contained pus; radical operation completed; Körner flap; eustachian tube curetted; no erosion of tegmen; sinus far back; exposed deliberately on account of high temperature, headache and chills; found healthy; wound packed and closed.

April 16. Temperature 99.4. No vomiting. Headache gone.

April 17. Temperature normal. Some pain in stomach; outer dressing changed, as it was soaked through.

April 30. Doing well; posterior wound was a little septic, but now healed; cavity clean; leaving hospital.

May 2. Had a bad headache today, which began last night; was a little chilly in the evening; no vomiting; ear clean; pulse 68; temperature subnormal; complained of blurred vision; very slight barely perceptible nystagmus to left (affected side). In the evening, pulse 76, temperature 98; no neck stiffness, no Kernig; arms very weak; hand grips weak but equal; can touch nose equally with both; abdominal reflexes normal; knee jerks active and equal; no clonus; plantar reflexes not elicited; bright mentally.

May 3. Vomited several times during night; constipation in spite of two purgatives; headache worse; felt behind eyes and back of neck; marked horizontal nystagmus to left. Dr. Courtenay examined eyes and reported blurring of the left disc and congestion of vessels; pupils equal and react actively to light and accommodation; no diplopia; slight slowness of left arm in rapid alternating movements; no Kernig; pulse 80, temperature 98.4. Cold peroxid in the ear during dressing produced nystagmus to opposite side and gave sensation of rotation. Readmitted to hospital.

Evening examination: Inclined to be drowsy all day and lies with eyes shut; no mental disturbance; rest position of eyes a little to right of middle line; a little horizontal nystagmus on extreme deviation to right; horizontal nystagmus to left. Pointing test with right arm perfect at shoulder, elbow, wrist and finger from above downwards; left arm fairly good but slight tendency to deviate to left from wrist; both grips equally weak; flaccidity very marked, both sides; does not touch nose very well with forefinger, slightly worse with left; no intention tremor nor misjudging of distance; some adiadochokinesis of left arm; on standing tends to fall backwards; unaffected by turning head. Cerebellar abscess was diagnosed, but it was decided to observe the case a little longer before operating.

May 4. Condition unchanged; lumbar puncture; fluid under considerable pressure; clear; report showed globulin not increased, 70 lymphocytes per cub. mm.; Fehling reduced.

May 5. Slept badly; headache rather worse; objective signs as before. At 6 p. m. had vomited four times since morning; headache much worse; great pain on tapping over left occipital region; decided to operate now. Operation 9:45 p. m.

Nitrous oxid anesthesia; posterior incision opened up; mastoid cavity healthy; bone extremely vascular; sinus uncovered freely; profuse hemorrhage with every bite of bone; cerebellar dura uncovered in front of sinus as far as possible without opening posterior semicircular canal, and also for some distance behind sinus, where mastoid emissary vein gave a lot of trouble; dura opened behind sinus, and brain immediately bulged out; cerebellum explored in a forward direction and sensation of going into a cavity felt, but no pus found. After operation patient said head felt better; nystagmus not so marked.

May 6. Vitality low today; no more vomiting; nystagmus less; some external squint, which is not paralytic, as both eyes can move. Headache still severe; patient apathetic and lies with eyes half open; in evening Cheyne-Stokes respiration; Dr. Sims saw patient in consultation; dura opened more widely behind and also in front of sinus; brain explored again; again sensation of going into cavity, but no pus; firm brain felt beyond; condition evidently localized encephalitis; hypertonic saline dressing; 10 cc. antistreptococcus serum given.

May 7. Still apathetic, but talks sensibly, though speech rather slurred; temperature subnormal, 97.3 during night; pulse 80; respirations 20; given 50 cc. antistreptococcus serum; in evening Cheyne-Stokes respiration very marked; came on fairly suddenly at 10 p. m. Intermissions of 30 seconds in breathing; given strychnin 1/30.

May 8. Had very good night and took fair amount of nourishment; at 7 a. m. breathing suddenly became intermittent; given strychnin and camphor; seen by me 10 minutes later; had revived somewhat, pulse and respirations better; forceps put on lateral sinus, which was divided, and dura opened widely; cerebellum bulged out; patient protested vigorously; he was quite conscious, though extremely apathetic and drowsy; squint as before; given 50 cc. serum; at 6 p. m. patient quite lively and asking questions; pulse now 102; temperature 98; breathing regular and natural for first time in days; taking lots of nourishment; beginning to object to nutrient enemas today; salt packs abstracting considerable fluid from wound.

May 9. Slept naturally during night; respirations more normal; patient much more lively mentally than even yesterday; objects greatly to dressings; very restless all day, and moving arms and legs about a great deal; still diplopia, though eyes nearly parallel; still nystagmus to left; left arm very flabby; right arm firm; both calves equally firm; left hand flops distinctly more than right on passive movement; pulse 88; still frontal headache; 50 cc. serum given.

May 10. Had a good night; brain pulsating well; forceps removed from dura; some odor from wound, probably from sloughs caused by forceps; temperature 100; still marked nystagmus to left; still diplopia, though squint not apparent.

May 11. Brain protrusion somewhat less; pulse and temperature normal.

May 12. Headache gone in evening.

May 13. Temperature dropped to 96.2 in early morning, but rose again; no headache; can move left hand fairly well with only a little intention tremor.

May 14. No headache; pulse and temperature normal; still some diplopia; brain protrusion same size, but firmer. Pointing tests: Pointing with left arm downwards from shoulder he deviated outwards, other joint movements normal; rest point of eyes now to left to middle line.

May 18. Progress good; feels well; no headache, but had a little the last two days. Brain bulging a little more; temperature 97. No diplopia; still nystagmus to both sides; white blood count 13,000.

May 20. Wound granulating well; salt pack discontinued; posterior pedunculated part of hernia about size of pigeon's egg.

May 23. Brain protrusion much smaller today; opening seen in anterior part, as though small abscess had burst. Brain pulsates better; pointing tests normal with both hands; touching nose not quite so good with left hand as with right; pulse and temperature normal.

May 25. Eyes examined by Dr. Courtenay; fundi absolutely normal; no engorgement of vessels; wasting and flabbiness of left arm now marked; adiadochokinesis marked.

June 2. Been sitting up for some days; hernia smaller; white blood count 7,000; grips of both hands much stronger; left apparently as strong as right.

June 16. Gets some headache on sitting up; hernia varies in size; pulse and temperature normal.

June 18. Sharp pain in head on sitting up; functional examination as before; white blood count 6,800.

June 21. Pulse and temperature normal; headache thought to be due to dragging of hernia on brain; hernia therefore pushed in with gentle pressure; dressing applied tightly.

June 24. No more headache or pain since 21st. Getting up.

June 27. Plastic operation under gas; flaps freed; scar tissue removed; edges brought together.

July 2. Went home today.

Further progress was uneventful. He reported on September 14, 1921; he was perfectly well and strong. There were no motor disturbances. The ear cavity was completely epithelialized and dry and the posterior scar scarcely noticeable. He was very anxious to be allowed to play football.

This case presents many interesting features. There was a definite history of injury, which possibly had something to do with the extension of the infective process to the brain. After the onset of the mastoiditis, symptoms suggestive of intracranial complications appeared. These were the severe headache, dizziness and vomiting. He had some chills, and, as the sinus was subsequently found to be healthy, these were probably due to the brain condition. The temperature also was much higher than is found in an uncomplicated mastoid, and one fully expected to find at least an extradural abscess and sinus thrombosis. There were no definite signs of cerebellar abscess when first seen, as is shown by the absence of nystagmus and adiadochokinesis, and the normal pointing test. The entire disappearance of symptoms after the mastoid operation and the latent period of two weeks were very misleading. At the end of this period headache, vomiting and nystagmus appeared. The nystagmus was significant of cerebellar involvement in being directed to the diseased side, when functional examination showed that the labyrinth was healthy. The eye grounds on the affected side showed slight changes. There was some incoordination and pointing error on the left side, and in fact most of the signs of a cerebellar lesion were present in a slight degree. The outward deviation of the left arm pointed to the anterolateral corner of the cerebellum, according to

Barany's localization, and this is just where the area of encephalitis was found. The edema apparently increased for some time after the second operation and would undoubtedly have led to death if the dura had not been widely opened up to relieve the pressure. From that point his recovery was slow but steady. The diplopia, which was evidently due to the pressure of the swollen cerebellum on the vulnerable sixth nerve, disappeared. The functional disturbances gradually diminished, and all went well except that the hernia would not go down. Later, when the patient complained of pain in the head whenever he sat up, as there was no sign of any increased pressure, it was thought that possibly the pedunculated hernia was being held by the ring of scar tissue round about, and could not get back inside the skull on that account. This turned out to be the case, as the brain tissue was reduced into the cranium with very little trouble, and the symptoms at once disappeared. The recovery from the wasting and loss of tone were remarkably rapid.

Case 2.—F. O., age 24, came to the office January 25, 1922, with a history of discharge from left ear since the spring of 1921. He had had growths removed from the ear in childhood, but the ear had not discharged in the interval. Two days previously he began to have pain in the left ear. Since this morning he had had a bad frontal headache. There was no dizziness or vomiting. He had never had any earache or headache before. No complaints in right ear.

Examination showed left ear full of pus, with a polypus in the roof of the meatus. The right drumhead showed a chalk patch; nose normal; left tonsil very large and red, right tonsil small; nasopharynx could not be seen; hearing, whisper, right, 18 feet; conversational voice, left, 16 feet; hears whisper one foot left with noise box in right; cold syringing left produced normal reaction. Polypus was removed with local anesthesia, revealing an attic perforation.

January 26. Headache and earache went away yesterday evening, but headache began again this morning and is now frontal and occipital; no vomiting; no chills; no pain in ear today; no mastoid tenderness; some pus in the left ear; tongue slightly furred; temperature 101.4; pulse 100; slight neck stiffness; Kernig sign present; right hand grip markedly weaker than left, although the patient is right handed; no aphasia; knee jerks sluggish, equal; no ankle clonus; pupils moderate and react actively to light; no nystagmus.

January 27. Patient slept well; still has frontal headache; vomited after salts last night; knee jerks active and equal; plantar reflexes flexor; temperature 101; pulse 66; bowels constipated; no pain in ear; discharge almost stopped; on syringing, fluid ran into nose; no tenderness on tapping skull.

January 29. Still has headache; pulse 56 after walking to office; temperature 96.8. Patient is rather dull mentally and slow in answering questions. Objective examination as before.

January 30. Admitted to hospital; mental condition brighter; no aphasia; pupils active and equal; no nystagmus; no diplopia; muscles flabby; tongue still furred; all reflexes normal, but right knee jerk slightly more active than left; still some trace of Kernig, though less than before; no neck stiffness; no headache today; coordination perfect; touches nose with both forefingers; pointing test normal with both hands; no loss of orientation of limbs; muscle sense good; no adiadochokinesia; cold syringing left produced rotary and horizontal nystagmus to right in fifteen seconds; pointed with both hands three inches to the left; pulse variable, 74 to 80, with pressure on eyeballs 56 and 70. Dr. Minnes examined eye grounds, but found no engorgement of the vessels and no papillitis; X-ray of skull showed nothing except some blurring of the mastoid cells; lumbar puncture gave opalescent fluid; no increase in pressure; examination showed opalescence to be due to contamination with blood; Fehling reduced, culture sterile; leucocyte count, 14,600; pulse still 54.

From the patient's dull mental state, infrequent pulse, subnormal temperature, headache, weakness of the right hand, increased leucocyte count as well as the chronic middle ear suppuration, an intracranial complication was diagnosed. From the fact that the disease appeared to be mainly in the attic and that the cerebellar functional tests were normal, it was thought that the condition was in the middle fossa. It was doubtful if an extradural abscess could account entirely for the condition unless it were of large size. It was probably a brain abscess. It was decided to operate and expose the dura of the middle fossa.

Operation: Cortex very thick and hard; lateral sinus far forward, healthy; on cutting down towards the antrum pus oozed out under pressure; mastoid process sclerotic with very few cells; antrum large and full of pus and cholesteatoma; tegmen apparently intact; radical mastoid completed; spur trimmed down and ossicles removed; cavity and tube curetted; tegmen removed and pus found outside the dura, which was white and sloughy; bone removed till healthy dura exposed; dura not pulsating, but no track discovered leading through it; Körner skin flap. It was decided to do no more at present, but to observe the patient for a few days, and if there was no improvement to explore the brain.

February 1. Slept badly last night; no vomiting; no headache; pulse 60.

February 2. No change; pulse in the sixties; right hand still rather weak.

February 3. Pulse in the seventies today; no headache; eyes examined by Dr. W. G. Fraser, who reported eye grounds normal and no limitation of visual fields nor alteration in the color fields. The culture from the mastoid pus gave a weak growth of streptococci.

February 4. Wound dressed, lower end septic, cavity clean; leucocyte count 17,000; lumbar puncture gave clear fluid, with no increase in tension; subsequent report on fluid, 120 lymphocytes per cub. mm.; Fehling reduced, cultures sterile.

February 17. Not much change in the last week; no headache, no vomiting, no pain, but patient always somewhat listless and apathetic; hand grips as before. In conversation it was discovered today that he could not remember what battalion he had been in in the army nor where he had been stationed. He could not remember the name of the village where he had been born, although he knew it was near London, England. He did not remember the name of the ship in which he crossed the Atlantic while in the Army. On the other hand, he remembered exactly how long he had been in hospital. He could not name a toothpick, but said it was for cleaning the mouth, similarly with keys, penknife, and other familiar objects. Eye grounds again examined, but showed no change.

February 18. Mental condition much the same as yesterday, but aphasia much less evident; could read and write a little, though not well, as education had been poor. No paresis of any of the cranial nerves; touch and pain felt equally well on both sides; taste and smell normal; right hand grip still weaker than left; power of legs equal; musculature poor; no loss of orientation of limbs; no astereognosis; Romberg negative; coordination perfect; leucocyte count 13,000; polymorphs 77 per cent.

February 22. Leucocyte count, 11,800; aphasia improved.

February 23. Said eyes felt funny today and that he saw double for a short time; vomited once in early morning; no headache; temperature 97.5; pulse 60. Clear mentally, but not good at finding words; no diplopia; convergence good.

February 25. Had a good day yesterday. This morning early had severe frontal headache; vomited; headache later passed off; been drowsy all morning.

February 27. No more vomiting or headache; leucocyte count, 13,000; eyes examined by Dr. Minnes, who reported retinal veins a little congested; a little diplopia at a distance, but impossible to say which muscle at fault.

It was felt from the first in this case that there was a temporosphenoidal abscess, yet after the extradural abscess had been drained his pulse and temperature returned to normal. Again, it was difficult to be sure that the paresis of the hand was a recent condition, as there was no change during the whole illness. When his wife was questioned as to his normal mental condition she stated that he had always been very quiet and silent and was rather dull. She had not noticed any change in him till within the last week. The aphasia was at times quite absent and was at no time very marked. The cerebrospinal fluid never showed any increase of pressure, although the cell count was increased. The only sign which was present throughout was the leucocytosis. However, in the last few days there had been the headache, vomiting and diplopia, so it was decided to explore the brain through the mastoid cavity.

March 1. Operation: Posterior wound opened up and cavity found to be clean and partially healed; dura of middle fossa bulging and not pulsating; incised and brain found loosely adherent; brain incised with bistoury, and at a depth of about one-eighth of an inch thin bluish pus was found;

about an ounce drained out slowly. The cavity had smooth walls and extended forwards about an inch, upwards and inwards about one-half inch, but not backwards at all. It took about 30 minutes for the cavity to empty. It was washed out very gently with saline and two tubes stitched in. The pulse and respiration were good throughout the operation. Later in the evening conscious, comfortable; pulse 90; temperature 99.

March 2. Occasional diplopia still; no headache, no vomiting; not a great deal of pus; more serous fluid.

March 4. Not so well today; looks pale; rather drowsy: pulse and temperature normal; abscess cavity not collapsed: not discharging as much as expected; in the evening pulse 69.

March 5. Restless through the night; vomited in early morning; complains of severe occipital pain; moaning and restless; temperature up to 102; dressed and thick pus found on dressings; about three-fourths of an ounce of thick pus pulsated out of brain, apparently from further forwards and further back than the original cavity; headache relieved immediately afterwards; leucocyte count, 25,200. In the afternoon he was semicomatose and groaning all afternoon; had a chill, and at 4 p. m. temperature rose to 104.2 and pulse 136. Died 5:50 p. m. He had had no convulsions.

Postmortem: Dura normal; convolutions on left parietal eminence slightly flattened; no exudate on vault; brain removed; firmly adherent round wound; some turbid exudate in interpeduncular space, none elsewhere; abscess cavity thick walled and leathery. Immediately outside the capsule there was an area where the brain was broken down and from which the thick pus had evidently come on the day of death: brain hardened before cutting. When the temporosphenoidal lobe was sliced a yellow soft area of encephalitis was seen to extend right up to the descending horn of the lateral ventricle, in which there was a little pus. There was none in the body of the lateral ventricle, though the fluid was turbid. Culture from the pus obtained at the last dressing gave staphylococcus albus.

This case presented many interesting points in diagnosis. He had had no headache except at the beginning and at the end of his illness. The only time he vomited during the earlier period of observation was after taking a strong dose of salts:

he only vomited again towards the end, when the encephalitis had begun to spread round the abscess capsule. The weakness of the right hand did not involve any of the other muscles of the limb. The aphasia was rather vague and transient, being only apparent for a day or two. As most of his symptoms gradually lessened one had considerable hesitation in exploring a brain which might have been only congested and gradually healing. However, after waiting some weeks without complete recovery it was felt that the time had come to explore. Arrangements were accordingly made to operate on the 20th of February . A neurologist was called in the day before to examine the patient, but on that day, as it happened, there was no aphasia, nor was there any other sign of brain abscess. He advised against operation, which was accordingly deferred in the meantime. However, a few days later more urgent symptoms appeared, and the operation was performed and the abscess drained. The fatal result was disappointing considering the encapsulation of the abscess, but was evidently due to the onset of encephalitis before the operation.

In conclusion, I should like to state my conviction that cases of brain abscess, though difficult to detect on account of the want of obvious symptoms, may be generally revealed by thorough and repeated neurologic examinations.

Taking the obscurity of the condition into consideration, we cannot afford to neglect any of the present diagnostic methods, but in every suspected case should subject the patient to an absolutely complete examination, including psychologic, neurologic, blood and cerebrospinal fluid examinations, etc. I would go further and state that every patient before undergoing a radical mastoid operation should have a neurologic examination.

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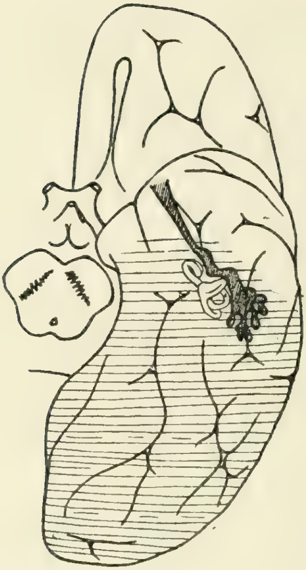

Fig. 1.

XLIII.

THE INTIMATE LYMPHATICS OF THE TRACHEA.*

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In a recently published and noteworthy series of studies on experimental lobar pneumonia by Blake and Cecil of the Army Medical School, there is among other strongly verified conclusions the statement that such pneumonia can only be produced experimentally by the direct instillation of the various types of pneumococcus into the trachea. They state that the typical lobar lesions cannot be brought on by spraying the nose and throat with virulent suspensions of pneumococci, but if the organisms are permitted access to the lower trachea, pneumonia results. The work was carried out on a large series of monkeys by injecting with sterile precautions broth suspensions through a fine needle inserted into the lumen of the trachea between the tracheal cartilages just below the larynx. The injections were made without producing coughing or gagging.

The principle involved in this work recalls a paper of Freudenthal's, published several years ago, discussing the treatment of intrathoracic conditions by intratracheal injections, in which the author offers some convincing proof that substances injected into the trachea do not pass beyond its lower limits. The same writer, in the discussion following the reading of Dr. T. W. Moore's article on intratracheal injections read before this society in 1919, said: "It is important to remember when treating the bronchi with this method that substances injected do not go down much below the level of the bifurcation."

In this connection the experiments of Coakley may also be recalled, in which after the injection of India ink solutions into the trachea of rabbits it was found after short periods of time, varying from fifteen minutes to two hours, that little or no pigment could be found in the trachea, bronchi or alveoli, having disappeared, the author states, by lymphatic absorption.

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These deductions—i. e., that suitable bacterial injections into the trachea produce lobar pneumonia,—that such injections (in doses not large enough to produce choking) probably never reach beyond the beginning of the primary bronchi,—and thirdly, that there is definite evidence of absorption in the mucous membrane of the tracheal wall,—these deductions very naturally awaken a degree of curiosity regarding the intimate lymphatics of the trachea, gratification of which is sought in vain among modern reference works. There appears to be available but few texts describing the actual lymph vessels of the wind pipe, in all instances volumes of Continental origin, and in none is the subject given more than a brief paragraph. In the divisions on the lymphatics in the larger systems of anatomy there is no mention made of the lymph vessels; text and illustrations are limited solely to a brief exposition of the tracheal lymphatic glands. The special works on the subject refer without exception to the difficulty of injecting the mucosa of the tube for determination of the distribution of the smaller vessels.

Periodical literature is devoid of any contributions to the subject, with the possible exception of the work of Winternitz, Smith and Robinson, on the rôle of the tracheal mucous membrane in the pathogenesis of pneumonia—and in this paper details are conspicuously absent.

Laryngologists seem to have neglected the matter entirely, and on account of its relation and importance to the ever increasing field of tracheobronchoscopy, I venture to bring some observations before you.

II.

In the fifth century B. C., Hippocrates referring to his discovery of a "white blood stream" and of vessels which take up nourishment from the bowel, was probably the first writer in history to mention the lymphatic system. Within three hundred years of his time we read of observations by Aristotle of peculiar vessels containing a colorless fluid, to be found accompanying certain groups of veins and nerves. Later there is recorded similar teachings by Erasistratus, Herophilus and Galen. This fragmentary knowledge was lost during the passage of medieval ages, and it was not until the middle of the seventeenth century, when Asellius of Pavia discovered the chyle vessels in animals, that the subject stimulated early modern anatomists. Following this demonstration there was a rapid succession of discoveries. The chyle vessels were found in man; Pecquet and Van Horne dissected the thoracic duct and cysterna chyli; lymphatics in the breast were isolated, and lymphatic trunks were demonstrated individually in various parts of the body. The eighteenth century marks the beginning of our modern conception, for at this time injection of the lymphatic trunks was introduced, and it was shortly demonstrated that the chyle stream and peripheral lymphatics were parts of a single general system.

The early efforts with injections were coarse and unsatisfactory. Air, milk and various colored solutions were put into the larger lymphatic trunks under pressure, the ramifying branches being followed for varying distances. Much of our knowledge regarding the gross lymphatics results from the injection of mercury, a method introduced by Anton Nuck and used consistently by anatomists for a long period. The method was a difficult one, however, and only successful in the hands of a few trained men. The technic for which Fohmann is largely responsible, namely, block or parenchymatous injections, was welcomed by workers in the anatomic field; by merely plunging the syringe into blocks of tissue or into the parenchyma of organs many small vessels could be filled and traced. When, later, a pressure apparatus was substituted for the syringe larger fields could be injected and further topographic advances were made.

The method of Gerota, presented in 1896, created a new era in the development of lymphatic investigation. Gerota recommended that the tissues be injected through a fine glass canula under pressure with a mixture consisting of Berlin blue, turpentine and ether. This technic, endorsed by Poirer, Most and other authorities, permits the tracing of the injected matter over greatly enlarged fields down to the smallest capillaries, and the collecting trunks can be traced to the receiving lymph nodes.

It is only since the work of Von Recklinghausen, who discovered that the lymph spaces and lymph vessels were lined with endothelial cells, that we possess a somewhat modern conception of their structure. Their embryologic origin has been in doubt until almost the last decade. Sabin has determined that the first lymphatic elements to appear in the developing ovum are budding processes from the endothelial lining of the veins, which by innumerable branching processes invade the body tissues down to the ultimate capillaries. It has been conclusively shown that the progress of the advancing capillary is by ameboid cellular outgrowths of the growing endothelial cells (E. R. Clark), and that actual growth is accomplished by cell division in the endothelial wall. The first endothelial bud sprouts out from the anterior cardinal vein to form the anterior lymph sac. An increasing series of buds unite to form plexuses which develop into additional sacs—"lymph hearts"—and from these organs continually advancing capillaries spread into the the developing fetal tissues.

Accounting for the peculiar characteristic anastomosis of lymph elements, it is interesting to note that investigators observing these advancing vessels under the microscope have demonstrated that the growing lymphatic tip has the remarkable chemicophysical power of avoiding blood capillaries while having an attractive affinity for lymphatic vessels.

The lymph glands have two sources of origin, according to embryologists: (1) A primary group formed from the embryonal lymph sacs by the bridging of numerous bands of connective tissue covered with endothelial cells. These nodes are found in chains lying along the course of the larger and earlier formed blood vessels. (2) A secondary group developing along the course of the larger lymph vessels by the formation of plexuses into sinuses and the subsequent bridging of the sinuses with lymph cords—follicles forming in the resulting trabeculæ. These glands are found in the developed body with no apparent relation to preformed venous structures.

III.

The difficulties encountered in the injection of the smaller lymph vessels of the trachea are several in number. The mucous membrane is firmly adherent to the subjacent cartilaginous rings which are imbedded in a strong dense fibroelastic membrane, and in comparison to the mucous lining of the nose is remarkably thin. Great difficulty has been experienced in attempting the injections in consequence of this firm, tense attachment. With injections carried out on the cadaver the principal objection has been the rapid destruction of the thin mucous surface due to evaporation and autodigestion. In the experimental injections made in living animals, after exposure of the trachea by open operation, results have been unsatisfactory because of the escape of the injected fluid into the tissues and because the operative procedure introduces the foreign substance into artificially opened lymph spaces and vessels, producing innumerable artifacts and errors which render subsequent deductions questionable.

It has occurred to me recently that probably many of the difficulties might be overcome by endoscopal injections of a proper substance into the tracheal mucous membrane of living animals with examination of the resulting absorption at varying periods postmortem. This method would obviate the necessity of injuring the windpipe and peritracheal tissues and, without interfering with the normal physiologic conditions and functions of the organ, would permit investigation of the real network of origin of the lymphatics in its mucous membrane.

Dogs were chosen for most of the present experiments, on account of the more favorable diameter of the lumen of the trachea, rabbits proving too small for satisfactory endoscopic work. Young dogs were found in preliminary investigations to lend themselves more readily to the work than adults, it being noticed in many instances that the mucous membrane was less firmly attached to the inner surfaces of the cartilaginous rings than in older animals. It was also remarked that though the normally thin membrane was if anything more thin in younger tracheas, they possessed, on the other hand, the valuable asset of greater elasticity, the membrane of adult animals proving very friable with small submucous injections. Thin dogs were considered to be the most desirable, as it was possible to control the position and movements of the trachea externally with the hand, and short necked animals were found to allow more exacting control during the actual injecting, as they permitted shorter shanked needles to be used with consequent better leverage.

The following individual methods of technic were devised and carried out with varying degrees of success:

(1) The animals were anesthetized with ether after the administration hypodermatically of morphin, and the Brüning electroscope was passed into the trachea. Injections were made with a Record syringe fitted with suitable sizes of the long shanked needles used at one time by Dr. Lynch and possibly still used by him for injections into the larynx. It was found to be essential that the needle point be rather short and of fine caliber in order to prevent laceration of the mucous membrane, and this danger was further guarded against by having the needle point bent into its shank at an angle of about fifteen degrees, thus allowing introduction of the point approximately parallel with the surface of the membrane. As a precaution against too great a pressure of fluid from the syringe the plunger was discarded and a small rubber bulb was capped over the open end. In this way the injection was more readily controlled, the rate of flow being easily regulated by gentle pressure of the fingers on the bulb.

The first injected substance used consisted of a thoroughly agitated 25 per cent solution of fresh India ink. It seemed advisable to dilute the solution, the stock ink being apparently too thick and oily to pass readily through the minute lymph vessels. Small quantities of the suspension varying from one-tenth to one-quarter of a minim were used after the first injection, larger amounts rupturing the mucous membrane with a spattering of the black fluid over the surface of the trachea. The ink injections proved to be of only partial value, as was to be expected. The lymphatic network could only be determined in small areas immediately surrounding the point of injection and only after almost immediate autopsy. In autopsies made eighteen to twenty-four hours after injection the ink was found only at the point of needle entry. In some of these cases, however, we were able to demonstrate large collecting vessels outside of the trachea by reason of engorgment with carbon bearing lymphocytes; and in all the cases carbon deposits were found by microscopic examination in the glands of the recurrent chain and deep internal jugular nodes.

(2) The same technic was carried out and a far more satisfactory demonstration of the lymphatic network was obtained by substituting for the India ink an aqueous solution of Berlin blue. This stain, which has a special predilection for endothelial cells was made according to the following formula:

Berlin blue	1 part
Oxalic acid	1 part
Water, distilled	12 parts

In autopsies made within two hours after injection the lymph vessels show up as a fine blue radiating network, the details of which are visible to the naked eye and very clearly defined with an ordinary reading lens.

(3) The experiment was repeated using a one-half of 1 per cent solution of silver nitrate after the method of Golgi. The method was discarded after a few trials, as the medium permeated the tissues indiscriminately, and after exposure to sunlight stained the mucous membrane more like a contrast stain than a selective one.

(4) Intratracheal injections of India ink were made through a needle plunged into the trachea between two of its upper rings in a manner similar to the method used by Blake and Cecil for the introduction of pneumococci into the trachea. In these cases no discoloring of the lymphatic vessels could be determined at autopsy three hours later, but it was evident that some lymphatic absorption had occurred, as carbon particles were found on microscopic examination of the peritracheal lymph nodes. Animals treated in this way were kept for a time under close postoperative observation, and in nearly all instances black streaks were apparent in the mucous secretion from the mouth after the first half hour. It is therefore probable that a very large factor in the disappearance of inert

substances injected into the lumen of the trachea is not absorption but the normal physiologic action of the ciliated epithelium of the tracheal mucous membrane.

(5) This technic was repeated, substituting Berlin blue for India ink. There was no macroscopic evidence of absorption and none could be determined under the microscope.

(6) In an effort made to secure a larger field of injection the endotracheal method described above (2) was repeated with modifications. After introduction of the electroscope a fine dry needle was inserted externally through the tissues of the neck into the tracheal lumen. After localizing the position of the point with the eye the needle was withdrawn from the lumen and, guided endoscopically, was reinserted into the lateral wall. This maneuver permitted the use of a longer needle point, the axis being parallel with the interspace, and there was consequently less danger of back flow along the course of the needle track.

(7) In conjunction with the latter method, more through accident than by design, a large quantity of the blue stain was injected into the peritracheal tissues, and postmortem examination was made in one case after one hour, and in a second at twenty-four hours. In the first instance there was a general flooding and staining of all the membranous structures, the dye having spread upward to a short distance from the occipital bone and downward to the bifurcation—guided by the fascial planes. The second case (after twenty-four hours) showed a blue tinted discoloration at the needle puncture, but the only evidence of flooding of the peritracheal spaces was a faintly green tinge in the membranes. The probable explanation of this phenomenon is that the stain, passing through the lymph spaces, entered the lymphatic vessels and, attacking the nuclei of the endothelium, caused a subsequent desquamation, the liberated cells carrying off the foreign matter.

In general the injections were made in groups of three at two levels; three in the upper trachea within two centimeters of the cricoid ring, and three just above the bifurcation. Each set consisted of one in the anterior wall, one in the lateral wall, and one posterior injection, the latter being made in the membranous strip between the open portion of the tracheal rings.

It must be stated in fairness to the reader that only a small proportion of the many injections made proved of value. In some instances no traces of the supposedly injected fluid could be found, due probably to an overzealous attempt to inject very minute quantities to avoid rupture of the vessels; and many times it was found postmortem (especially before a short needle was used) that the point had penetrated the tracheal wall and emptied its contents into the loose areolar tissue of the peritracheal spaces. In many cases the needle, however gently manipulated, tore through the delicate mucous membrane and the stain was spattered freely into the lumen of the trachea. Even in many of the successful experiments the limitations of the various methods used were apparent. The lymphatic course could be followed in most instances for only a relatively short distance from any one point of injection, as far as the superficial network was concerned, though it was possible to illustrate the continuous routes of absorption in many of the experiments, connecting the tracheal efferents with their receiving nodes in the general lymphatic system.

IV.

A study of the findings in a large number of tracheal injections indicates:

(1) That there are practically no lymph vessels other than the fine intercellular lymph spaces in the mucous membrane where it passes over and is attached to the cartilaginous rings.

(2) The tendency of lymph flow in this network of origin on the surface of the cartilages is upward and downward to the nearest interspaces.

(3) When the injected fluid reaches the interspaces (or, as most frequently occurred, was injected directly into the interspaces), the course of absorption becomes horizontal, running into small vessels lying between the rings. These tiny trunks vary from one to three in number, usually single in the cervical portion and increasing at lower levels of the tube.

(4) The lower ring surfaces are somewhat more easily injected and the fluid shows a tendency to spread with equal ease to the next upper or lower interspace. Higher in the trachea there is a distinct tendency for the absorbed pigment

to migrate upward to the first neighboring interspace rather than downward. It is also noted at the central portions of the tube that the superficial network is consistently less dense than at either the upper or lower levels.

(5) In the anterior wall injections the fluid crosses the midline with ease and shows an equal tendency to spread horizontally to both lateral walls, with a marked inclination to circumscribe the tube.

(6) The lateral injections, and this holds for both upper and lower levels, may spread anteriorly, but the characteristic tendency is posteriorly to reach the membranous portion of the trachea.

(7) The posterior injections into the membraneous wall demonstrate the presence of a large number of lymph vessels, and larger individual vessels at any given level compared to the number and size of the vascular elements in the cartilaginous portion of the tube. Here also is apparently a greater tendency to upward and downward communication, the incomplete cartilages offering no obstruction here. In the pars membranacea, it may also be demonstrated, the lymphatic vessels are more numerous and of larger caliber in the thoracic portion compared to higher levels.

(8) The horizontal collecting vessels are more numerous in the lower interspaces and seem to increase in caliber in the interspaces near the bifurcation. They are joined in their course by minute collectors from the surface of the two adjacent rings, and the general direction of their flow is toward the posterior membranous wall. There is a disappearance of the majority of these trunks beyond the open ends of the cartilaginous rings in a radiating network of fine vessels which cover a comparatively large area in the membranous wall—a network which usually extends across the midline to similar vessels on the opposite side. It is here, according to Poirer, Most, and a few other investigators, that the efferents leave the tracheal wall to join the general lymphatic system.

(9) Though there is a vast anastomosis of these vessels in the peritracheal connective tissue between the windpipe and the esophagus, by far the greater portion of the main trunks join the lymph vessels accompanying the recurrent laryngeal

nerve. These large collectors are interrupted in their course by nodes—the paratracheal glands—corresponding to the recurrent chain in man. From these nodes drainage to the deeper internal jugular groups can be traced by the deposits of the carbon particles of the India ink.

CONCLUSIONS.

There is a partial lymphatic absorption of inert substances by the tracheal mucous membrane in a normal uninjured and uninflamed state.

The cartilage rings, dividing the superficial network of origin into segments, offer successive lines of defense against lymphatic spread of tracheal infection.

Absorption from the mucous membrane when no inflammatory condition exists is less active in the middle third of the trachea than in either the subcricoid area or that portion approaching the bifurcation.

The tendency of lymph drainage at any given segment is to the posterior wall of the trachea, through which offending matter is carried from the field to neighboring lymph nodes.

Though the lymphatic function is segmented by the tracheal rings, and in this way may be considered regional, nevertheless anastomosis between the various levels is free by way of the plexuses in the posterior wall of the tube. There is also, to a very minor degree, an intersegmental drainage by way of individual capillaries which at irregular intervals hurdle the belly of the cartilages from interspace to interspace.

Hypothetically substituting the pneumococci for carbon particles and stains, pneumonia may be caused by absorption of the organisms through the mucous membrane of the trachea into the recurrent nodes and jugular chain, arriving at the lungs by way of the lymphatic ducts, great veins, right heart and pulmonary artery.

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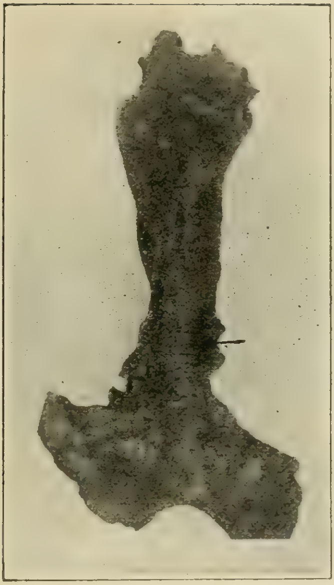

Fig. 1.

Trachea of young dog; endoscopal submucous injection of India ink. Autopsy in one hour. Arrow points to site of injection. Note the enlarged black capillaries in the interspaces.

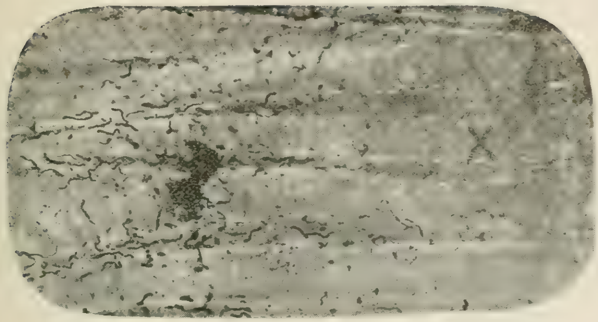

Fig. 2.

Trachea of young dog; endoscopal submucous injection of India ink. Autopsy in two hours. Magnification: 6 diam. (X marks anterior wall.) Shows tendency of the pigment to be carried to the interspaces and toward the membranous part of the tube. The specimen is is taken from the subcricoid area.

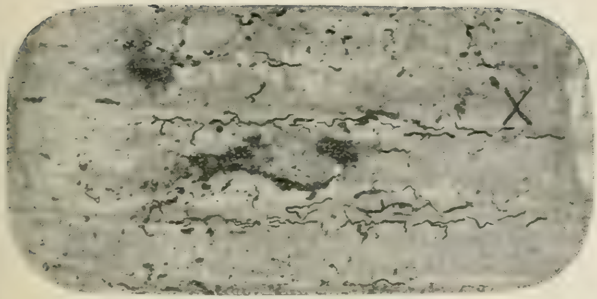

Fig. 3.

Trachea of dog; submucous injection of India ink. Autopsy in one hour. Magnification: 6 diam. Notice here the marked tendency of the pigment to pass through lymph vessels in the interspaces and toward "X," which marks the direction of the posterior wall. The specimen is taken from the suprabifurcation area.

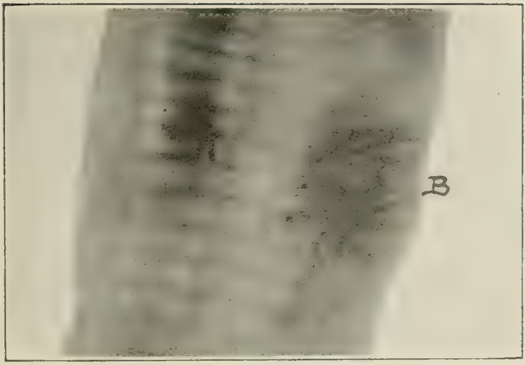

Fig. 4.

Trachea of young dog; submucous injection of Berlin Blue. Autopsy in one hour. Actual size. "A" marks the point of injection and at "B" is seen the stained network of the membranous portion of the wall through which the horizontal collecting vessels become extratracheal.

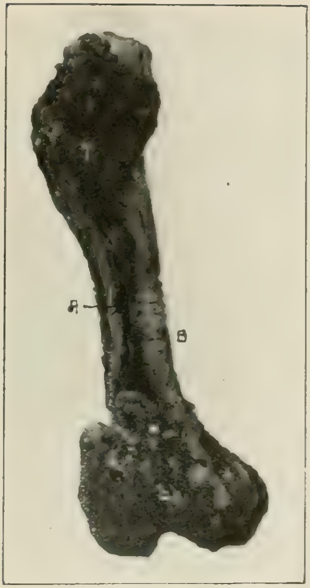

Fig. 5.

Trachea of young dog; submucous injection of India ink. Autopsy in three hours. "B" marks the point of injection. "A" indicates the lymphatic cords accompanying the recurrent laryngeal nerve—engorged with carbon bearing lymphocytes.

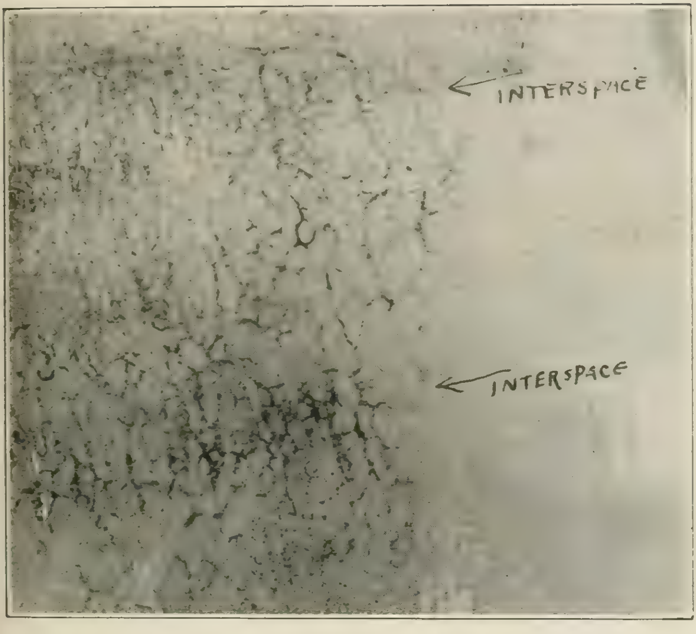

Fig. 6.

Trachea of young dog; submucous injection of Berlin Blue. Autopsy in 30 minutes. Magnification: 16 diam. Showing the rich dense network of lymphatics near the bifurcation. Here also is seen the tendency to absorption through the interspaces, the general direction being from the point of injection to the posterior wall.

XLIV.

MALIGNANT TUMOR OF THE TEMPORAL BONE.

By Alexander Zebrowski, M. D.,

New York.

In the period of twenty years of practice limited to the diseases of the ear, nose and throat, I have had opportunity to observe and operate upon three cases of malignant tumor of the ear. The first case, sarcoma, I described in 1908 in the American Archives of Otology. The second case, cancer of the temporal bone, I treated from nearly the very beginning of the disease. This case was published in 1912 in the Monatschrift für Ohrenheilkunde.

Two years ago, when in the military service, I performed a radical mastoid operation upon a Polish soldier, with chronic left side otitis media complicated by mastoiditis. Four weeks after the operation the facial palsy, exuberant granulations in the wound and the general condition of the patient became so suspicious that a microscopic investigation of the granulations was made. This investigation proved that we had to deal with a case of malignant tumor.

In all three cases mentioned above a thorough macro and microscopic postmortem examination was performed.

It is well known that the malignant tumors of the ear are exceedingly rare. Even a very busy practitioner may have no opportunity in the years of his practice to observe a single case. In the large ear clinics these cases come to view sometimes, but their ratio to the other ear diseases is very small. Bezold in his well known records of 20,000 ear patients treated in the space of 24 years had only four carcinoma cases. Sarcoma of the ear occurs still more rarely. In my article cited above, I have found in the accessible literature only three cases (two described by W. Milligan and one by Oppikofer), published from 1896 till 1908. All cases of the sarcoma of all parts of the ear (auricle, external, middle and internal ear), published till 1896, are mentioned in the work of Asch,[1] and the sum of them is less than 100.

The difficulty of picking out from literature all cases of some rare form of disease like the malignant tumors of the ear is evident from the article of Dr. O. Benesi,[2] published recently in the Monatschrift für Ohrenheilkunde, 1920, No. 11. The author affirms that the metastatic carcinoma of the ear has not been described yet ("Krebsmetastasen am Ohre sind in der Literatur nicht beschriben," l. c. page 961). Meanwhile we find in the very interesting work of Dr. Schlichter[3] four articles (Habermann, Alt, Neuman, Schwabach) mentioned, which deal with metastatic cancer of the ear!

Historia morbi of my case is very short and simple. In June, 1919, a soldier, 22 years of age, small but well nourished, was admitted to the ear department of Ujazdow Military Hospital in Warsaw. Internal organs (heart, lungs, kidneys, a. s. o.) normal. Since childhood purulent discharge from the left ear. The right ear, nose and throat normal.

The examination of the left ear revealed: Profuse purulent discharge, drum membrane absent, the drum cavity filled with granulations, red in color, slightly bleeding on touch.

The hearing power diminished—whisper, 2 meters, Rinne negative, Weber lateralized to the left ear. Caloric reaction positive. A common treatment was applied, and the patient was informed that he would be rejected on account of his ear trouble.

Three weeks later the patient's temperature rose (37.6° C.) and he complained of some pain in the ear and in the mastoid region. The mastoid process was slightly tender on pressure, the discharge became profuse and offensive in odor. This condition lasted some days and the patient was offered an operation.

On June 28, 1919, the radical mastoid operation was performed. In the mastoid process I found a small quantity of pus and granulations. The antrum was well developed and also filled with pus and granulations, as well as the tympanic cavity, which were thoroughly curetted. The ossicles (mallet and incus) were absent. The stapes was imbedded in the granulations, which were carefully removed. The cranial fossæ were not opened. Plastic after Panse method.

The course of the usual after-treatment was very unusual. The wound behind the ear was filled with a large quantity of pus and had to be reopened. The granulations were pale and exuberant; the general condition of the patient became worse. A month after the operation a sudden paresis of the left facial nerve appeared and that made me suspect that we had to deal here with a malignant tumor. The evening temperature, which immediately after the operation became normal, rose again to 38.0° C. With the double curette some particles of granulating tissue in the wound were removed and a microscopic examination made. In all preparations the same picture appeared.

Among the stroma built from the connective tissue infiltrated here and there with leucocytes we find thrown epithelial herds. In some places we can observe the junction between these herds and the covering epithelium.

The herds are of different shapes, round, elongated or branching irregularly. The cells constituting these herds show numerous abnormalities, differing from the normal epithelial cells. They are of various sizes and various shapes. The size of their nuclei is different—some nuclei are stained stronger than others. Besides this the cells show many degenerations as vacuolization and cornification, which is most distinct in the middle of the herds. (Fig. 5.)

According to this microscopic investigation, we diagnosed a cancroid, whose starting point was probably an epidermis of the external ear channel.

A similar result was obtained from a microscopic postmortem examination.

Taught by the two cases observed earlier, I called the relatives of the patient to let them know that the condition of the poor man was hopeles. The patient refused a second operation. Meanwhile the X-ray treatment had been applied (ten applications every second day—there was no radium in the hospital at that time). The treatment was without effect. On the contrary, I had the impression that the growth of the tumor was even more rapid. Two months after the operation the condition of the patient was as follows: (Fig. 1.) Total paralysis of all branches of the left facial nerve. The left ear absolutely deaf. Caloric reaction negative. The tumor behind the ear had begun to disintegrate. The tympanic glands in the mastoid region and on the neck were swollen and enlarged—in short, it was a typical picture of a malignant tumor of the temporal bone, which I had had the sad experience to observe already twice before.

Three and one-half months after the operation the wound looked like a large, deep hole, with the walls covered with pale abundant masses of tumor and pus. (Fig. 2.) As we see on this figure the auricle was held in situ by a relatively thin piece of healthy skin. Some weeks later this piece was destroyed by the disintegrated tumor masses and the auricle fell off at the change of dressing. (Fig. 3.) Constant headache appeared and grew more severe every day—a sign that the tumor had involved the dura. Morphin was administered and it was necessary to make many injections day and night. General cachexia which could be noticed already on the second month after the operation developed and was the cause of the exitus on December 18. The patient was in the hospital about six months.

Postmortem examination showed following changes:

Exhausted, emaciated body of a young man. No pathologic changes in the internal organs; no metastases. In the region of the left ear a great, deep wound involving a space between the zygoma and occipital bone. Practically the whole temporal bone, except a part of the pyramid, was destroyed and in place of it we found masses of tumor. Left auricle was absent. After removal of the roof of the skull and of the brain it was evident that the external surface of the dura in the region of the temporal bone was covered with tumor masses which did not penetrate through the dura into the cranial cavity. The brain was anemic, but without any changes. After removal of the dura it was seen that the tumor masses had destroyed not only the remnants of the mastoid process (after the radical operation) and the middle ear, but also the part of the pyramid. The part of the pyramid which remained (Fig. 4) had the appearance of a small process, covered with a thin layer of the tumor mass penetrating the bone.

The lymphatic glands on the neck were enlarged, but only in the immediate neighborhood of the destroyed temporal bone. The lymphatic glands on the other side were normal.

As we see from the postmortem examination, in this case we had to deal with the malignant tumor limited to the left temporal bone without any metastases, involvement of the brain, as was described in other similar cases. The only cause of the exitus was cachexia, as in my case of the carcinoma cited above. In the case described by Danziger,[4] the tumor masses, after destroying the left temporal bone, penetrated through the dura into the cerebrum, where in the left temporal lobe they formed a large growth. This growth caused aphasia and was the immediate cause of the exitus. In my first case of sarcoma, cited above, the tumor masses destroyed the processus odontoideus of the second vertebra, which was broken when the patient sat up in bed. The processus odontoideus entered into the cranial cavity and injured the medulla oblongata, causing the sudden death of the patient. Usually, as we see from the literature, the common cause of the exitus in these cases is a cachexia.

How should we operate a case of a malignant tumor of the temporal bone, if we operate at all? In all three cases upon which I operated, a radical mastoid operation was made, which proved unsuccessful and obviously insufficient. As we see in the literature on the subject, there is only one case, that of Jansen,[5] in which eight months after the radical operation no relapse had appeared. In the article of Grasser[6] a very interesting case is described so far as that a diagnosis was made by paracentesis of a reddened and bulging drum membrane. Through the opening in the drum, masses of malignant growth protruded. In this case, after operation there was no relapse in the space of eight years. About the malignant tumors of the ear Doyen in his "Traité thérapeutique chirurgicale," Vol. II, writes that it is impossible to operate upon such cases: "Ces néoplasmes sont au dessus des ressources de chirurgie"; "l'opération radicale est impossible dans l'état actuel de la chirurgie."

I think that an early operation, consisting of total resection of the whole temporal bone, including the pyramid, may be successful. In the case described above, nature shows us the way to follow when operating. Despite such an immense destruction of the part of the skull, during all the time not even the slightest bleeding occurred, although the tumor laid open all the big blood vessels (carotid artery, sigmoid and petrosal sinuses, bulb of the jugular and jugular vein). The early facial paralysis, the most prominent symptom of the malignant tumor of the ear, teaches us that when operating we must sacrifice this nerve. The involvement of the pyramid shows that we must resect the whole pyramid up to the internal ear channel.

Such an extensive operation is not only technically feasible, but, as I know, has been performed three times, once with successful result.[7] The indications for these operations were twice a destruction of the pyramid by a bullet; once the operation was performed accidentally on account of necrosis of the pyramid (Ruttin, cited in the article of H. Frey.[8]) In the interesting article of H. Frey, the author describes briefly the method of the total resection of the pyramid and says that such an operation can be indicated above all in the cases of malignant tumors of the temporal bone. The technic proposed by H. Frey consists in short in a radical mastoid operation, after which the bony attachment of the pyramis should be cut away and then the pyramid removed in toto.

My experience, limited to the performing of this operation on many specimens, has taught me that the most difficult step in this operation is the laying free of the dura, which must unavoidably be injured when removing such a large block of bone as a pyramid. We can work without injuring the dura only in one manner, namely, by chiseling carefully and removing every splint of the bone after a previous thorough separation of the dura (Fig. 6).

I should propose in a case of early recognized malignant tumor of the temporal bone such an operative measure. (The particulars I will describe in a special article.)

A. Preliminary operation.

1. Thorough radical mastoid operation. The dura of both cranial fossæ must be laid free as far as possible.

2. Ligation of the carotid artery and of the jugular vein. Removal of lymphatic glands.

B. Some days later the resection of the temporal bone.

1. Blocking of the sinus by using the method of Whiting-Meyer.

2. Laying free the facial nerve, which will serve us as a guide to the internal ear channel.

3. Removal of the posterior part of the pyramid until the vestibulum and internal ear channel have been reached.

4. Removal of the upper parts of the pyramid. Cave sinus petrosus sup.

5. Removal of the cochlea and of the inferior and anterior parts of the long external auditory canal and of the drum cavity. The opening of the eustachian tube is laid free.

6. Resection of the facial nerve and removing of the bone in which the facial canal is located.

7. Total resection of the auricle with the tragus and cartilaginous portion of the external ear channel.

On the figure 6 we see the specimen with the operation made.

I believe that such an extensive operation, when performed early, can be successful, as the total resection of the larynx in the cases of laryngeal malignant tumors. The facial paresis can be corrected by means of an anastomosis with the hypoglossal nerve, which should be made some weeks later.

When we bear in mind that the prognosis in cases of malignant tumors of the temporal bone is absolutely unfavorable, it is clear that only such an extensive operation, with removal of all parts in which the tumor can grow, may be employed when we intend to operate upon such cases at all. Every other operative measure will always prove insufficient and cause only the more rapid course of the disease.

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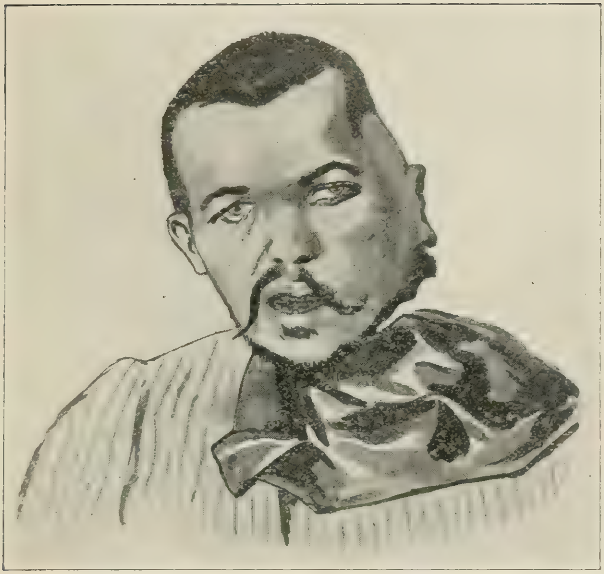


Fig. 1.

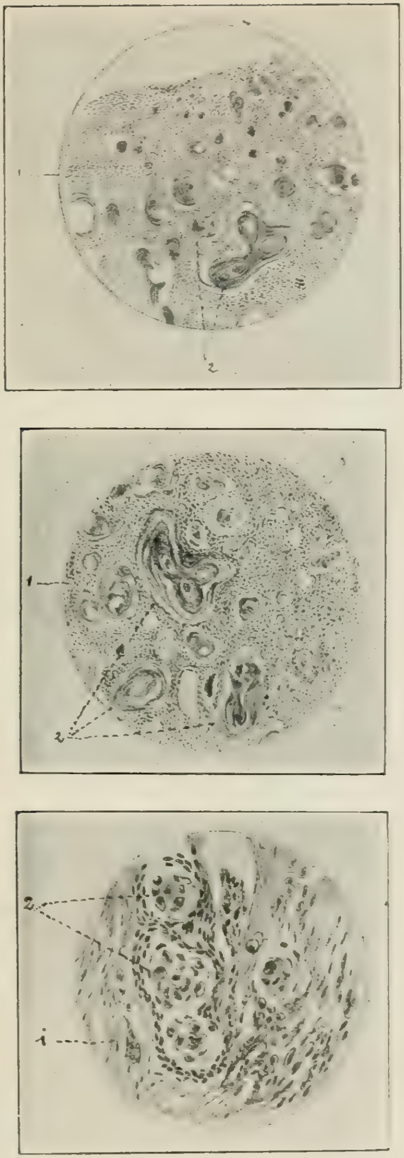


Fig. 2.

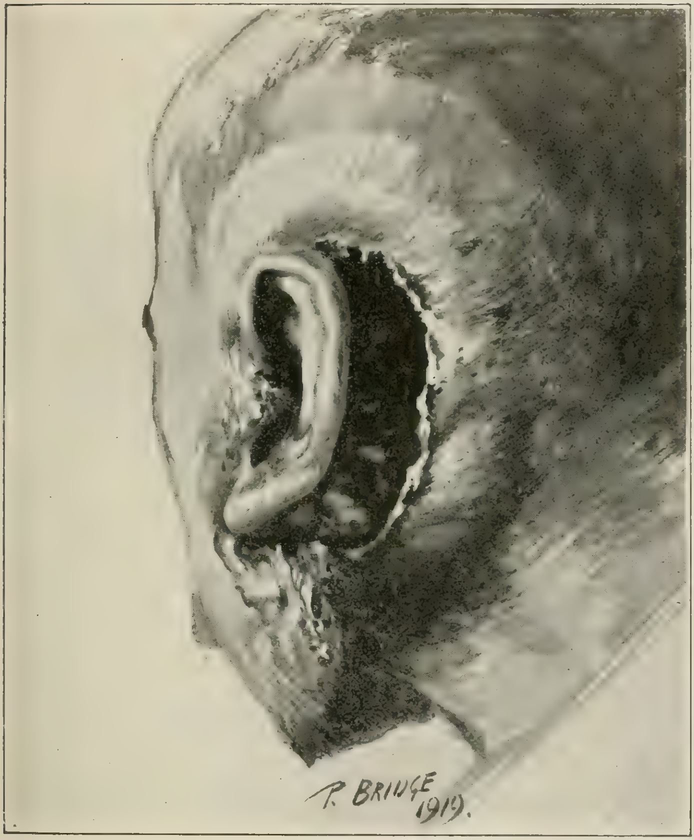


Fig. 3.

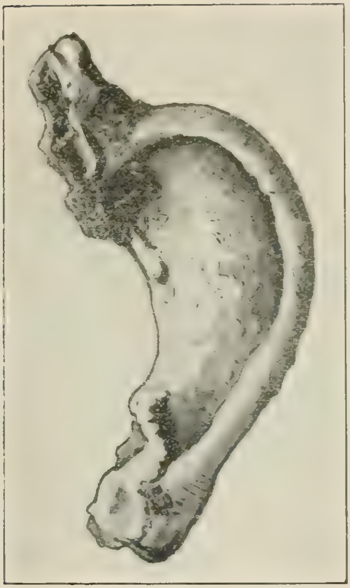


Fig. 4.

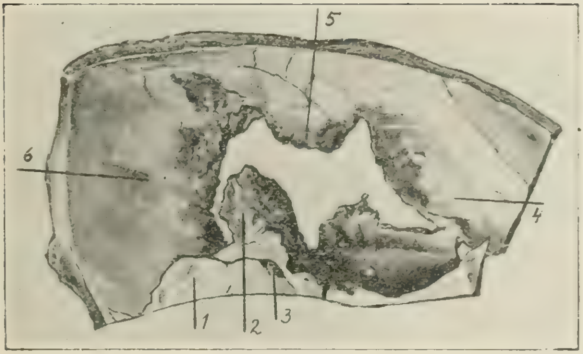


Fig. 5.

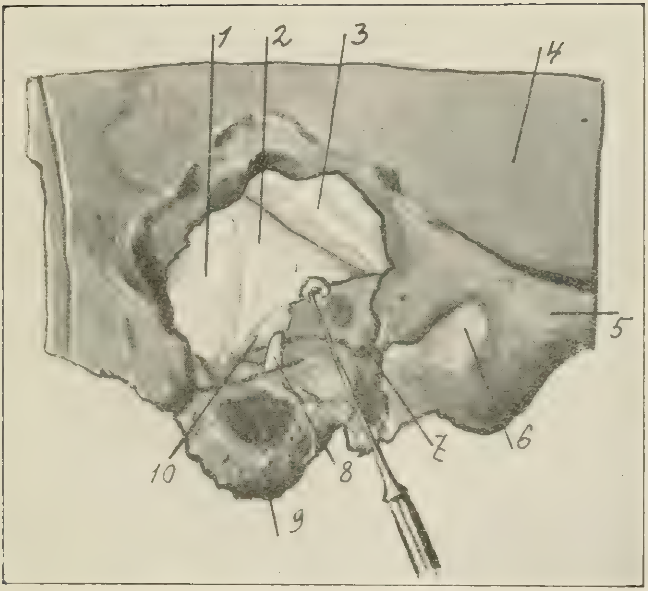


Fig. 6.

XLV.

A BRIEF REVIEW OF THE ANATOMY AND SURGERY OF THE TEMPORAL BONE, WITH REFERENCE TO THE MASTOID IN HEALTH AND DISEASE.

By J. C. Keeler, M. D.,

Philadelphia.

I am prompted to select the mastoid for the subject of study because of its interesting development into the temporal bone; its susceptibility to infection; its unsuspected etiology in some of the more serious intracranial complications and its closely allied clinical manifestations of other diseases which frequently are deceptive to the physician.

All students of medicine, as well as the surgeon, frankly acknowledge that it is most difficult to comprehend and to clearly visualize the anatomy of the temporal bone and the vital structure within and adjacent to the mastoid, during the process of dissection or operation.

In view of these facts and the urgent necessity for a thorough familiarity with the delicate anatomic structure of the mastoid, liable to irreparable damage during operation, I shall present photographs of original dissections of the temporal bone and of the mastoid, descriptive of their beginning in the fetal stage, trace their early development in the quite young and continue until it becomes an integral of the temporal bone in the matured adult.

In utere, the periotic capsule is cartilaginous. Ossifying material is not deposited until the end of the sixth month. This is done in four centers, according to their relation to the capsule. The epiotic is the last to appear, and is first seen at the posterior part of the posterior semicircular canal; and from this center the mastoid process takes its rise and that is the only center to be given consideration in this paper.

In the new-born there is a complete absence of the mastoid as is seen in Figures 1, 2, 3 and 4. The first suggestion of such a process presents itself as a small tubercle, behind the upper part of the pars tympanica. That part of the petrous bone from which the mastoid process develops presents a surface so flat as merely to suggest a prominence. The development of the cell space in the mastoid proceeds by cellular proliferation. Beginning at the posterior superior periphery of the antrum, it advances towards the transverse sinus.

The mastoid antrum is the only pneumatic space present in the temporal bone in the new born and is larger than that of the adult. The mastoid antrum opens into the tympanic attic, a small chamber lying in the upper and back part of the tympanum. Its size varies with age; it is present in birth and increases until about the third or fourth year when it gradually diminishes, owing to the encroachment of the bone developed around it.

The squamo mastoid suture lies between the anterior surface of the tuberculum mastoidum and the posterior portion of the pars squamosa and forms the external wall of the mastoid antrum in the new born as shown in Figure 4. It gradually disappears during the first years of life, although it may persist in the adult as a jagged, deep furrow on the external surface of the mastoid process. In such case it may be described as representing the appearance of a depressed fracture.

The roof of these chambers is formed in part from the squamous and in part from the petrous shown in Figure 2 (a) and (c). The suture passes directly through the tegmen tympani and antri on the inner surface at the junction of the petrous and the squamous, Figure 2 (a) and (c). It is quite patulent and for this reason susceptible to meningeal symptoms in case of acute suppuration of the middle ear.

The outer surface of the temporal bone in the new born lacks the osseous external meatus seen in the adult. The membranous meatus is connected with the shallow rim of bone, Figure 1 (b). The pars tympanica to which the membrana tympani is attached is shown in the groove, Figure 2 (b). It is the close relationship between these membrances that makes a child sensitive to pain upon the manipulation of the auricle during an attack of acute otitis media. Owing to the patulent condition of the pars squamosa and pars petrosa, Figure 4 (a) and (c), the escaping pus in an acute suppurative otitis media causes postauricular swelling so frequently seen in the very young.

The outer wall of the antrum is formed by a plate descending from the squamous, Figures 1 (a) and 2 (a). This wall is very thin in early life, but is developed by deposits from the periosteum. The depth of the antrum increases from the surface.

The roof of the antrum, tegmen antri, is merely the backward continuation of the tympanum. The level of the roof is indicated by the posterior root of the zygoma. The level of the floor of the adult skull at the tegmen antri is, on an average, less than one-fourth of an inch above the external osseous meatus.

In the development of the temporal bone after birth, the two conspicuous changes brought about are the formation of a mastoid process and of a bony external meatus, or rather the development of the tympanic plate of the temporal bone. During the first year of life this tubercle takes a downward course. This is caused partly by its own growth and partly by the action of the muscles. The mastoid process does not assume its adult form until the third year of life (Figure 5).

In some cases the mastoid is massively developed; in others, reduced to a short protuberance. The cells may be of the pneumatic or of the diploetic type, or they may be mixed in form. In fact, innumerable varieties exist. The pneumatic cells of the mastoid process are lined with a delicate, gland-less membrane, a continuation of the mucous membrane of the tympanic cavity.

The antrum communicates with the lower vertical cells of the mastoid through its floor. The floor being on a lower level than the opening into the tympanum, drainage of the antrum is difficult and the fluid readily filters into the lower cells.

The bend of the sigmoid part of the lateral sinus (Figures 8 b, 8c and 29) with its descending portion is seen behind the mastoid antrum and cells. A vertical line drawn one-fourth of an inch behind the posterior bony wall of the external auditory meatus and between the levels of the floor and the roof will indicate, in the majority of cases the position of the anterior convexity of the sigmoid sinus in the adult.

If the mastoid cells be of the pneumatic type (Figures 26 and 32), the lateral sinus will usually be found deeply situated and more posterior, hence less liable to exposure or injury; whereas, in the diploetic type (Figures 8a and 29) of cells, the location of the sinus is invariably reverse. This has proved characteristic in chronic purulent inflammation as revealed in the radical mastoid operation. It is not uncommon to find that the posterior osseous wall of the auditory canal forms the anterior wall of the sigmoid portion of the sinus and is frequently exposed or injured at that point. The lower and vertical cells of the mastoid are developed later than those of the antrum and vary in their contents. Only about 20 per cent contain air. The foramen, an opening for the emissary vein (Figures 5a) is on the posterior border of the mastoid process.

I consider roentgenography one of the most valuable single aids in the study of the mastoid in health and disease. Experience and great skill are required to interpret the shadows of the picture; but that test ought to be a part in the study of every case in determining whether or not to open the mastoid. Any abnormality in the development of the mastoid; the type and extent of the cell area; the character of the osseous structure; the position of the lateral sinus; or any chronic or acute necrotic erosion of the mastoid and its adjacent structure will be revealed by the roentgenogram.

The accompanying radiograms well serve to illustrate those different conditions. Figure 25 is that of a physician who had an acute purulent mastoiditis with necrotic erosion revealing an extradural abscess. Undoubtedly, the cells extending over the zygoma were of the type shown by Figures 8, 8b, 13, 20, 23 and 24.

Humanity owes to the scientific medical profession an everlasting gratitude for the vaccines and serum therapy. Diphtheria and typhoid fever are no longer numbered among the chief predisposing causes of middle ear infection with mastoid complications. Statistics prove measles, scarlet fever, pneumonia and influenza fruitful sources of middle ear infection. Let us hope a serum to prevent or annul the virulence of those diseases will soon be discovered.

Great precaution should be taken to protect the ears when swimming and diving, lest water enter the middle ear by the way of the eustachian tube or by a patulent ear drum and superinduce mastoid involvement or intracranial complication. This is especially likely if the ear drum be patulous from an existing otorrhea or if cholesteatoma be present. Infection of the middle ear demands prompt surgery. Often a minor operation in the early stage of inflammation will prevent serious complications and a major operation. Any inflammatory process of the middle ear becomes at once a surgical case.

Reviewing my clinical experiences, I recall with surprise the frequency of mastoid infection following tonsillectomy and submucous resections, and the utter indifference patients manifested concerning diseases of the ear. A very great majority of patients coming to our clinics may be grouped in four classes:

1. Those suffering acute pain.
2. Those with acute otorrhea, entirely oblivious of the thought of complications. More than 50 per cent of this class cannot give an intelligent history of the onset of the disease; its cause; its duration or its treatment.
3. Those who are rapidly becoming deaf; others who have vertigo, and those threatened with or perhaps have developed intracranial complications.
4. Those who have lost the function of hearing and are

being outstripped in their vocations by others not thus handicapped.

In a series of two hundred and fifty mastoid operations, there seemed to be no age limit; I have operated on a three months' old infant and on a patient seventy-two years of age.

The right or the left mastoid was involved about an equal number of times; also, neither the acute nor the chronic type of mastoid prevailed, the number of operations in both cases being about equal. That in the acute form of the disease, the streptococcus and the pneumococcus were the predominating type of infection; in the chronic cases there was a mixed variety. In one case of chronic mastoiditis complicated with septic sinus thrombosis the colon bacillus was found.

Operations were performed in a few cases complicated with pneumonia, tuberculosis, the exanthematous diseases and diabetes. In the diabetic case, 10 per cent sugar was found in the urine when admitted to the hospital. As this was an emergency, operation was done under local anaesthesia, using Schlech's fluid; in another case a radical mastoid operation was done under morphine and scopolamine, socalled Twilight sleep. Gas anaesthesia was used in the tubercular case.

The facial nerve due to necrotic erosion of the fallopian canal (Figures 31 and 32) was found exposed at the time of the operation. There was no preexisting facial paralysis, nor did paralysis ensue; whereas, if the facial nerve be exposed or injured during the operation, paralysis of the face is most likely to occur.

The frequent necrotic exposure of the dura with a moderate degree of localized thickening or extradural abscess shows that nature will in some instances check the invading organisms by a protective barrier. The sigmoid sinus passing through the mastoid as it does is in great danger of infection by necrotic erosion. Here again, nature protects the vessel wall by a barrier of granulations. Septic sinus thrombosis does occur, however, and death is inevitable, unless prompt surgical relief is instituted.

It is the consensus of opinion that the best preventive of chronic otorrhea is opening the mastoid before intracranial complications ensue. I cannot recall a single case of mastoid disease, either the acute or chronic, where there were no complications and death ensued, that might not have been averted if the operation had been performed early.

Drooping of the posterior wall of the external auditory canal is regarded as pathognomic of mastoid involvement by some authorities; others equally well informed claim this is not an infallible indication for operation. This is undoubtedly true in that type of case where pus is confined in the cells of the tip or lower portion of the mastoid. In such cases operation is as much indicated as if all the classical signs were present. Be obedient, therefore, to the homely adage, "Wherever there be pus, let it out."

Textbooks enumerate pain, tenderness, redness and swelling over the mastoid as the classical symptoms of mastoid involvement. Usually those symptoms are present; but their obsence does not prove that the mastoid is not seriously involved. For instance, a mastoid with a thick sclerosed cortex never shows those well defined symptoms because the internal resistance is less than that in the external direction.

I have seen patients with a very extensive necrosis and in a general septic condition, with practically a normal temperature and with no tenderness over the mastoid even upon very hard pressure, who proved upon operation to have had a perisinus or an extradural abscess, or both. If the mastoid had not been opened intracranial complications would have resulted, without doubt.

I believe the hearing has been preserved in many cases of acute infection of the middle ear and the mastoid when the operation has been performed early. In chronic otorrhea, the hearing may or may not have been impaired as much depends upon the character of the involvement of the middle and internal ear. Loss of hearing is likely to follow operation because much of the structures employed in the conduction of sound waves has been removed. But in my experience the end results have been gratifying.

ILLUSTRATIONS.

FIGURES 1, 2, 3 AND 4

represent dissections of the temporal bone in the fetal stage.

Figure 1 presents the three portions of the fetal temporal bone:

(a) The pars squamosa which is the thin porous shell-like bone, the lower portion forming the outer wall of the epitympanum.

(b) The pars petrosa which is detached from the pars squamosa and pars tympanica. It presents the inner wall of the tympanum, the upper half representing the recessus epitympanicum and the antrium.

The lower half presents the recessus mesotympanicum. The promontory is readily seen. The fenestra vestibula with the stapes inserted is in the superior posterior portion, and a few millimeters below, is seen the fenestra cochleae which opens into the first turn of the cochlea. It is closed with a membrane called the membrana secunda and it separates the tympanum from the scala tympani.

FIGURE 2—A, B AND C.

Figure 2. This is the inner surface of (a) the pars squamosa with (b) the pars tympanica attached, which represents an incomplete ring with a groove for the insertion of the membrana tympani.

The malleus and the incus are held in position by part of their ligamentous attachments. The head and neck of the malleus and the body of the incus repose in the recessus epitympanicus. The stapes is detached from the process of the incus and is located in the fenestra vestibuli as described in Figure 1 (c).

The outer wall of the recessus epitympanicus ad antri is formed by the articulating surfaces of the pars squamosa and the pars petrosa.

FIGURE 3

Presents the articulation of (b) pars tympanicus to (C1) pars petrosa.

The malleus, the incus and the stapes are held in position by their ligamentous attachments. The fenestra rotunda is seen on the inner central border of the pars tympanica and the stylomastoid foramen for the exit of the facial nerve is on the outer posterior border, while in front and below, is shown the foramen for the carotid artery.

C2 presents an unobstructed view of the fenestra vestibula and the fenestra cochlea; the promontory; the tympanic end of the eustachian canal and the groove immediately above to which is attached the tensor tympanic muscle.

Above the fenestra vestibuli is seen the osseus capsule of the horizontal semicircular canal; above and posterior to this, is the antrum.

FIGURE 4

Shows three sections, (a) the pars squamosa; (b) the pars tympanica; (c) the pars petrosa of the fetal temporal bone have united. The Rivinian notch (d) is clearly defined. The ossicles are held in position by their ligamentous attachments; (e) the petrosquamosal articulation is revealed by the fissure on the posterior border; (f(the stylomastoid foramen is seen on the posterior border of the pars tympanica; (g) the carotid foramen is at the anterior inferior border; (h) the eustachian foramen is at the apex; (i) the Glaserian fissure is clearly shown.

The extremely porous condition of the bone in the fetal stage is noted.

FIGURE 5—A, B, C AND D,

Represent the temporal bone with the early development of the mastoid in the child.

Figure 5 (A) shows the early development of the mastoid in a child. Below the tip is the depression of the digastric groove. Between the tip of the mastoid and the posterior border of the pars tympanica is the stylomastoid foramen for the exit of the facial nerve. The dark notch immediately below is the jugular bulb. Upon opening the mastoid, greatest care must be taken not to sever either the nerve or the jugular bulb. The notch on the posterior border of the mastoid is the foramen through which passes the mastoid emissary vein.

The pars tympani has developed to a considerable size and forms the tympanic plate in later life. The Glaserian fissure is the depression between the pars tympanica and the articulation of the condyloid joint.

Figure 5 (B). This gives a view of the pars petrosa as the bone is lying on its concave surface.

(C). The large foramen is the carotid canal.

(D). The small foramen is the stylomastoid for the exit of the facial nerve, and (E) the depression between them is the jugular bulb. This again shows how readily the nerve and the jugular bulb may be severed. The mastoid is seen as a slight elevation.

Figure 5 (C) gives a view of the posterior surface of the petrous, the concave surface up. The deep groove of the lateral sinus is clearly seen ending at the jugular bulb, just posterior to the carotid foramen and is indicated by the large dark foramen. The superior and posterior semicircular canals are opened and are represented by the two dark lines above the jugular depression. The facial nerve is represented by a dark line entering the internal osseous meatus as seen in the upper part of the petrous, directly above the jugular foramen.

Figure 5 (D). Viewing the anterior convex surface of the temporal bone. The dark foramen is the continuation of the carotid canal. Next is the lamina spiralis exposed showing the course of the facial nerve as it passes over the first turn of the cochlea to enter the fallopian canal on the inner anterior wall of the tympanum. The top foramen is the exposure of the superior semicircular canal.

The tegumen tympani and antri are opened, exposing the incus and the head of the malleus.

The dark irregular surface just above the petrosa is the transverse sinus. The long slender lines with branches are the grooves for the middle meningeal artery on the inner surface of the squamosa.

FIGURE 6.

The fully developed temporal bone and mastoid (Figure 6) with its massive zygomatic bar and root. The squamosa, the petrous with the mastoid as its base, and the tympanic plate which has developed from the incomplete ring of the infant (Figures 1b, 2b and 4b).

The Glaserian fissure is posterior to the condyloid fossa; the silkworm gut projecting from the fissure represents the corda tympani nerve.

Macewen's triangle is formed above by the posterior root of the zygoma; the anterior boundary, by the posterior auditory osseous canal. The third or posterior line of the triangle is an imaginary line joining these two lines. This triangle was considered of great importance in former years in trepanning the mastoid to drain the purulent process, as it was the most direct approach to the antrum without injury to the lateral sinus.

When removing the cortex of the mastoid, the operator must never go above the posterior root of the zygoma lest he open into the middle fossa of the skull and injure the brain.

FIGURE 7.

The temporal bone divided through the petrous. On the left side is seen the ear drum, the incus and the malleus. The tensor tympanic muscle is shown attached to the malleus.

The corda tympani nerve is represented by a fine light line crossing the membrana tympani at the end of the process of the incus.

On the right, the stapes is seen in the fenestra ovalis with the stapedius muscle inserted in the neck of the stapes.

The osseous capsule of the horizontal semicircular canal is seen above the upper border of the nerve.

The dark lines on the promontory are the tympanic branches of the glosso-pharyngeal, or Jacobson's nerve.

The heavy dark line between the horizontal canal and the fenestra ovalis represents the course of the facial nerve.

Note the mixed type of mastoid cells.

FIGURE 8—A, B AND C.

Figure 8 (A). This represents the temporal bone with the cortex removed from the mastoid, showing the mixed type of cells, the diploetic predominating. The cells extend over the zygomatic bar as far as the tempomandibular articulation. Involvement of the cells in an acute purulent process of the mastoid would result, very probably, in intracranial involvement of the middle fossa. The dark line represents the corda tympani nerve passing through the Glaserian fissure.

Figure 25 X-ray plate shows an erosion of the squamosa. In this location an extradural abscess associated with acute suppurative mastoiditis was found.

A patient suffering from epileptic seizure of the Jacksonian type, with chronic purulent process of the mastoid involving the cells extending into the squamosa, resulting in an inflammatory irritation of the dura, recovered after a radical mastoid operation.

Figure 8 (B) shows the inner surface of Figure 8 (A). Note the cells of the mastoid extending into the squamosa. This indicates the likelihood of purulent inflammation with early meningeal irritation.

The facial nerve is represented by the dark line crossing over the modiolus and in front of the semicircular canals at the point where it enters the tympanum. The deep groove of the lateral sinus is clearly shown and is far forward.

Figure 8 (C). The cells extending over the squamosa are clearly defined. We may look for early intracranial complication in case of purulent infection of the mastoid in a patient with this type of cells. The lateral sinus and the semicircular canals are clearly shown.

The small dark spots in the petrous back of the internal osseus meatus represents the foramen for the entrance of the facial, vestibular and cochlear nerves.

The head of the malleus and the incus is seen in the epitympanicum.

FIGURE 9.

This is the anterior concave view of the petrosquamosal portion of the temporal bone. The dark lines on the convex surface of the squamosa are the grooves for the middle meningeal artery. The petrosa is opened showing the spiral of the cochlea. The semicircular canals and the epitympanicum show the articulation of the malleus and the incus. Note the thinness of the bone separating those structures from the middle fossa.

FIGURE 10.

This is the petrous with the squamous removed. The articulation of the ossicles within the tympanum are clearly seen. The dark half circular lines represent the superior and posterior semicircular canals.

Here again we have the pneumatic and the diploetic form of cells.

FIGURE 11

shows sections of the petrous bone and the spirals of the cochlea.

The internal ear is a complex chamber; the bony labyrinth contains the membranous labyrinth, and the specific receptors or sensory surfaces of the internal ear.

(b) The small section on the right side of the plate is the cochlea. It is interesting to note the califie deposits around the spiral. It would be of scientific interest to know whether or not this subject was deaf.

FIGURE 12—A AND B.

Figure 12 (a). The osseous capsule of the labyrinth.

(b) The upper foramen is the fenestra ovalis; the lower, is the fenestra rotunda. The silkworm gut represents the course of the facial nerve as it passes between the oval window and the horizontal canal.

(c) The internal osseous canal removed with the hope of showing the foramen for the passage of the facial, vestibular and cochlear nerves. The circle on the upper surface is the superior canal.

Figure 12 (b). This shows the facial nerve (7th) passing through the internal osseous auditory meatus over the first turn of the cochlea, under horizontal canal. The spirals of the cochlea are clearly defined.

(e) The three semicircular canals are clearly defined. They serve chiefly the function of equilibration. Under the semicircular canals can be seen the oval and round windows with the promontory.

FIGURE 26.

Surgery of the temporal bone consists chiefly of opening the mastoid to drain the suppurative process and to remove necrotic bone. When the infection has extended into the internal ear or labyrinth, it may be opened to prevent further extension to the meninges.

The cortex is removed and the antrum is opened. The pneumatic type of cell is well defined.

FIGURE 27

Is the simple mastoid operation.

The upper border of the mastoid is on a level with the posterior root of the zygoma. (Fig. 6.) The large cell over the digastric ridge extends into the petrosa, opening into the jugular bulb could easily occur unless the operator has a clear visualization of the anatomy.

FIGURE 28.

This shows the exceptional beveling of the outer wall of the epitympanicum and emphasizing the importance of opening the mastoid in the direction of the course of the external auditory osseous canal.

FIGURE 29.

The mastoid opened showing how high the cells extend into the root of the zygoma.

FIGURE 30

Shows the modified radical, the type of operation frequently selected in special cases in an effort to effect the cure of a chronic suppurative process by drainage of the mastoid, antrum and epitympanicum, yet preserving and retaining in position the ossicles and membrana tympani. Note how superficial and far forward the bone covering the sigmoid portion of the lateral sinus. The cells in this case were of the diploetic type.

FIGURE 31

Represents the radical mastoid operation. This is selected as the operation of choice in the chronic form of middle ear and mastoid infection to rid, completely, all necrotic and infected areas from this region. The tympanum, the antrum and the mastoid are converted into one large cavity. To accomplish this, it is necessary to remove the posterior part of the external osseous auditory canal, the facial nerve passes under it. (Fig. 32.) The fenestras ovalis and rotunda are clearly exposed. The pyramid showing the superior, posterior and external canals is seen above.

Note—To visualize clearly those structures lying within the danger zone, this dissection has been made more extensive than is usually performed on the living subject.

FIGURE 32.

The radical mastoid operation is extended in order to expose the semicircular canals and the course of the facial nerve, as it passes between the fenestra ovalis and the horizontal canal.

FIGURE 33

Shows the labyrinth opened. The instrument is shown passing into the cochlea under and back of the horizontal portion of the fallopian canal and protruding above the horizontal semicircular canal. The opening of the superior and posterior semicircular canals is seen just above the point of the instrument.

PLATE 1. FIGURE 12.

Partly sclerosed mastoid. A few small cells remain at upper posterior angle. Lateral sinus large, superficial, and far forward.

PLATE 2. FIGURE 13.

Small cell type of mastoid. Cell area very large, and extends to near angle of orbit. Normal.

PLATE 3. FIGURE 14.

Small cell type. Very small cell area. Normal.

PLATE 4. FIGURE 15.

Above—large cell type. Normal on both sides.

Below—A. Dense sclerosis. Lateral sinus well posterior.

B. Small cell type, normal.

PLATE 5. 7079R. FIGURE 16.

Type doubtful, either undeveloped or cells were obliterated early in life. Very little evidence of sclerosis. Lateral sinus fairly well posterior, but superficial.

PLATE 6. 7079L. FIGURE 17.

As compared with R, there is more evidence of sclerosis. There are a few cloudy cells toward the tip.

PLATE 7. 3284L. FIGURE 18.

Small cell type mastoid fairly large area. Cell shadows are cloudy. Slight sclerosis at tip. Lateral sinus rather far forward. 6881L shows this same mastoid 3½ years later. Note increase in sclerosis.

PLATE 8. 6881L. FIGURE 19.

Partial sclerosis with cloudiness of remaining cells. Note extent of cells posterior to lateral sinus which is rather far farward, sub-acute. Compare with 6881R, and also with 3284L, which is the same mastoid 3½ years previously.

PLATE 9. 6881R. FIGURE 20.

Small cell type, fairly large area. Cells extend forward to level with posterior border of temporomandibular articulation. Normal.

PLATE 10. 6220L. FIGURE 21.

Mixed cell type, small cell area. Normal.

PLATE 11. 6220R. FIGURE 22.

Sclerotic mastoid. Note bone defect in antrum region, and along anterior border of the lateral sinus toward the mastoid tip. Lateral sinus fairly forward and very superficial. Compare with 6220L for difference in appearance of groove for lateral sinus.

PLATE 12. 4001L. FIGURE 23.

Same cell area as shown on the right side. These cells are cloudy, showing less contrast. Earliest X-ray signs of acute mastoiditis.

PLATE 13. 4001R. FIGURE 24.

Small cell type, rather large cell area. Note cells extending forward over the zygoma. Normal. Compare with 4001L.

PLATE 14. 6912R. FIGURE 25.

Sclerotic mastoid, irregular, clouded cell structure posterior to antrum region and along anterior border of the lateral sinus toward the tip of the mastoid. Circular bone defect in skull about ⅝-inch in diameter. In the absence of history of operation this probably indicates a necrotic area. Lateral sinus fairly well anterior.

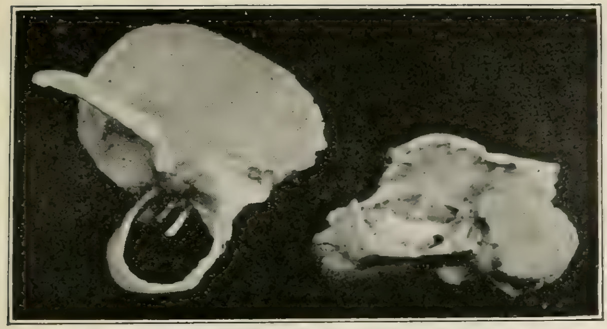


Figure 1.

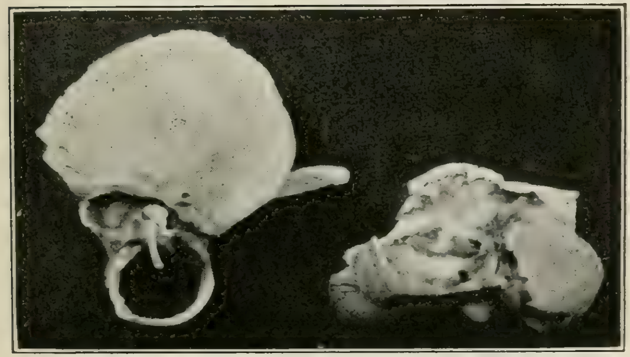


Figure 2.

Figure 3.

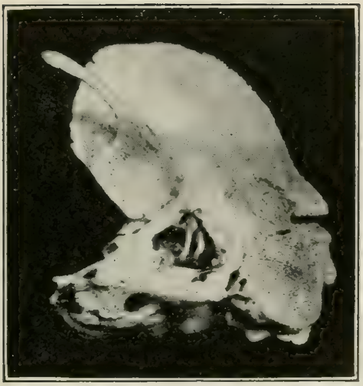


Figure 4.

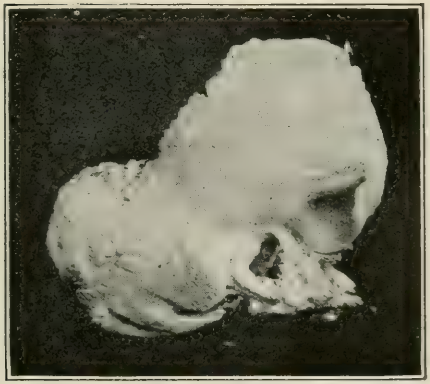
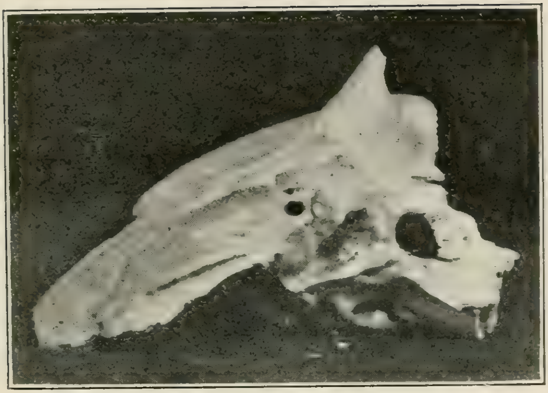


Figure 5 (A).



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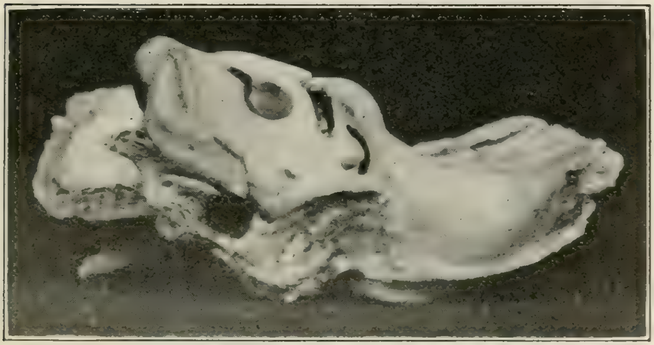

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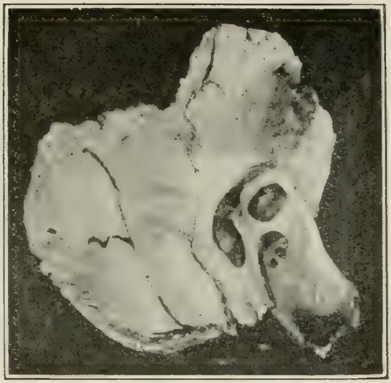

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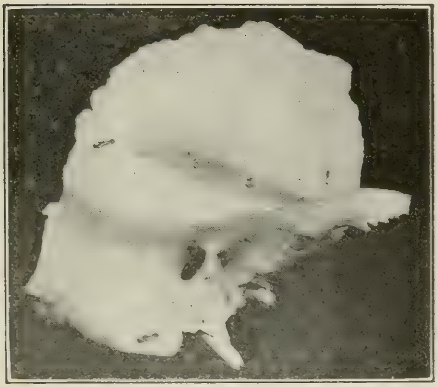


Figure 6.

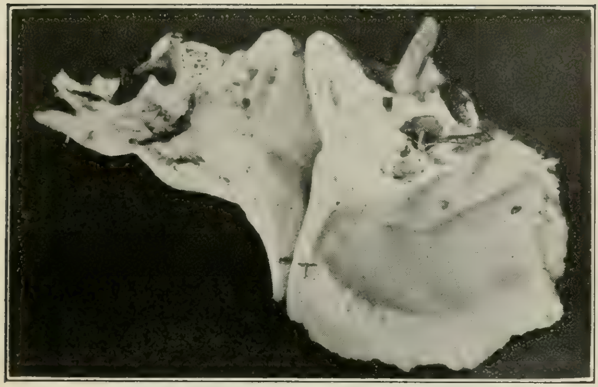


Figure 7.

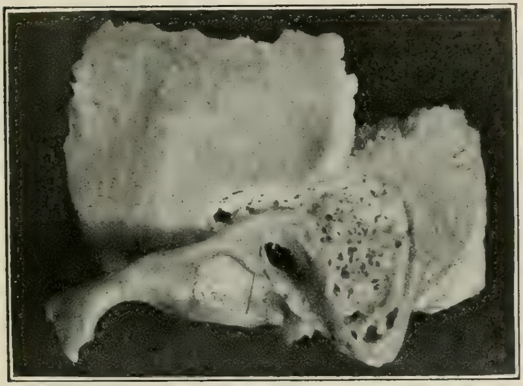
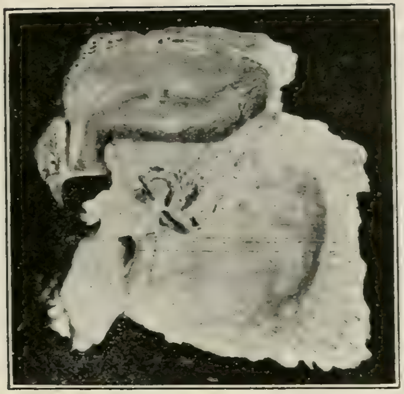


Figure 8 (A).



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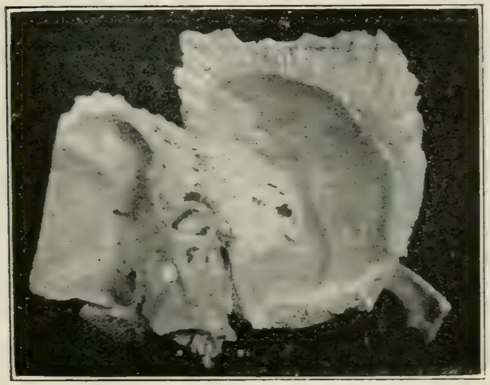

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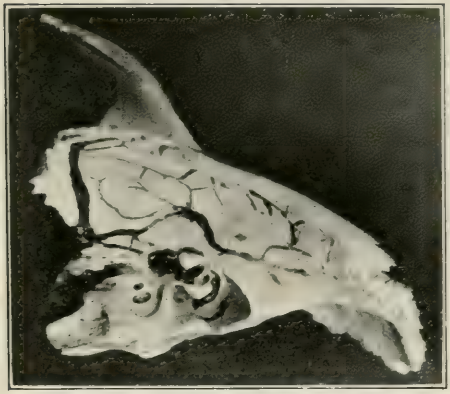

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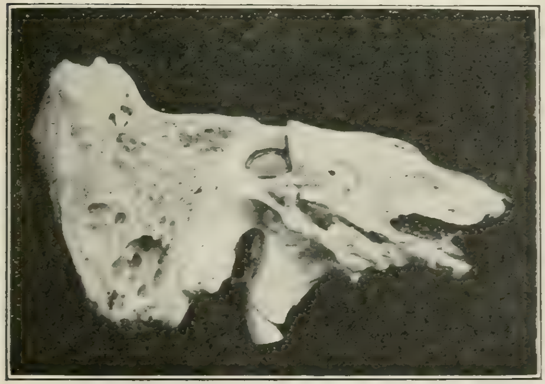
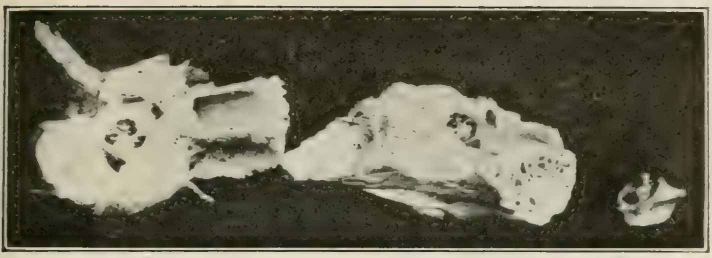


Figure 10.



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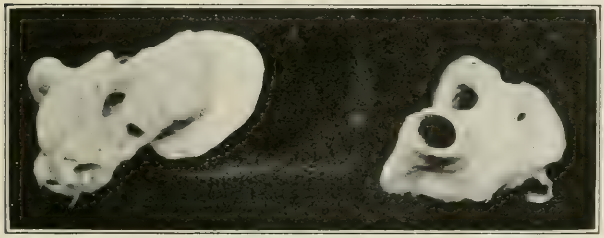

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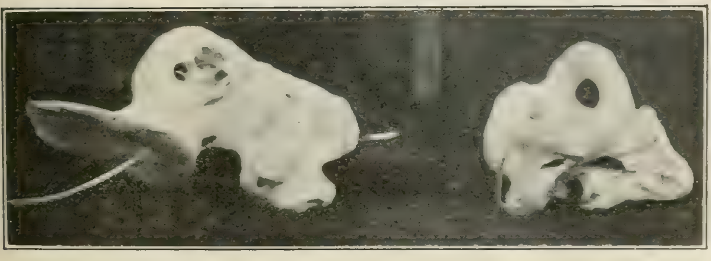

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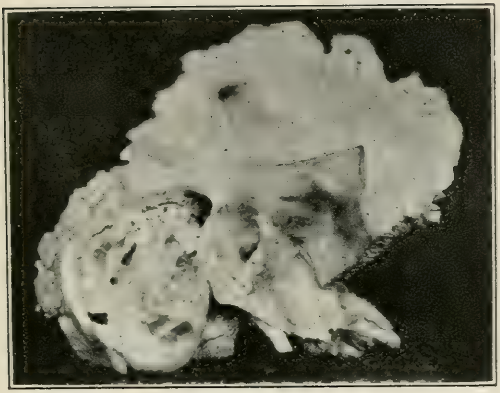

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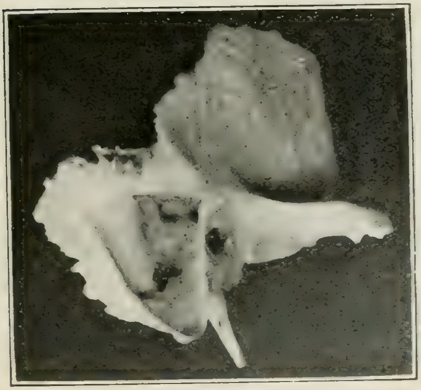

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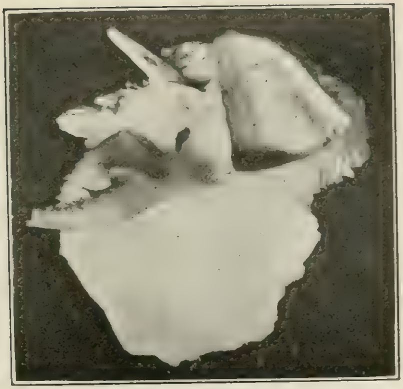

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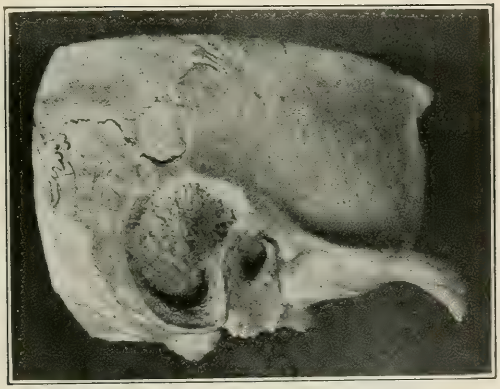

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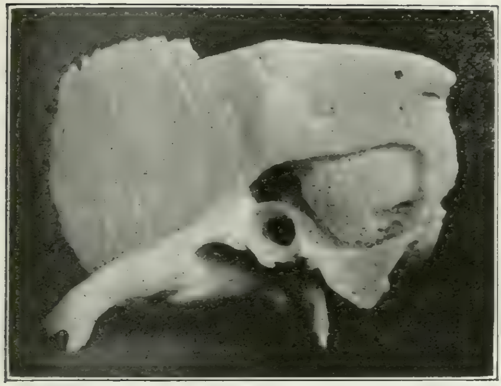

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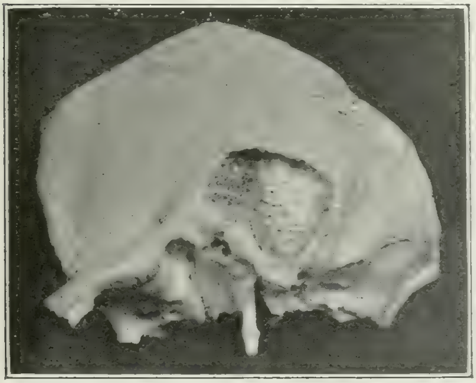

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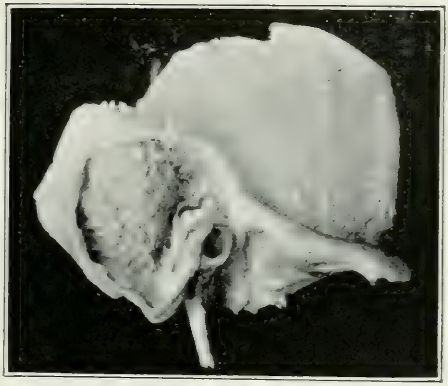

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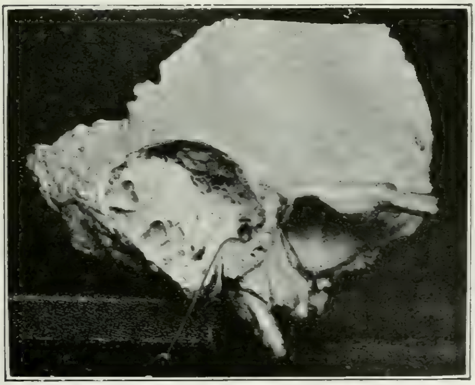

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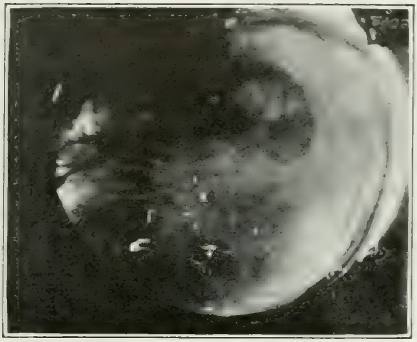
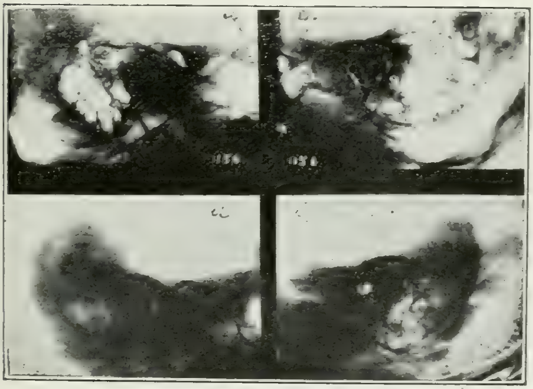


Plate 3. Figure 14.



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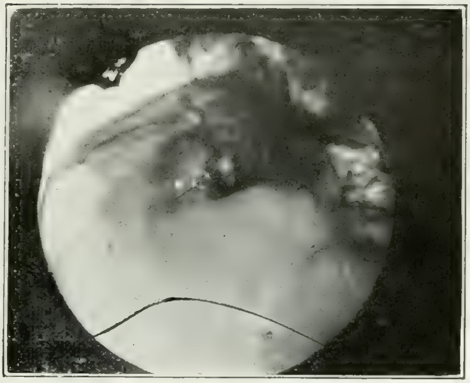
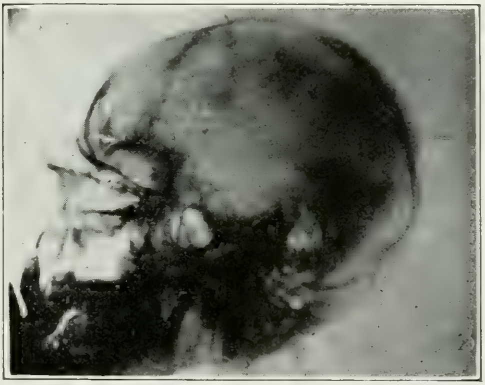


Plate 1. Figure 12.



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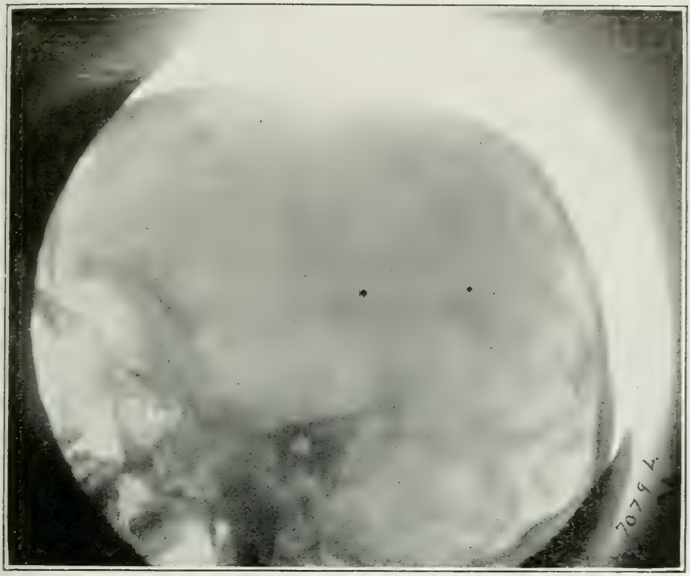

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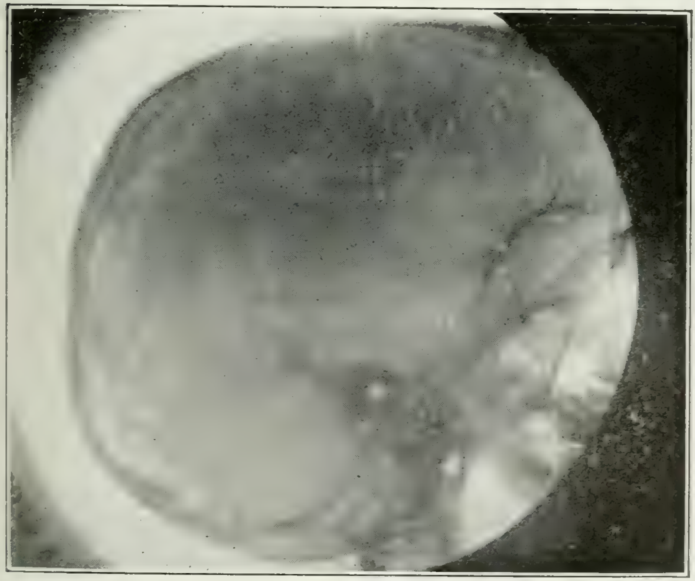


Plate 6. Figure 17.

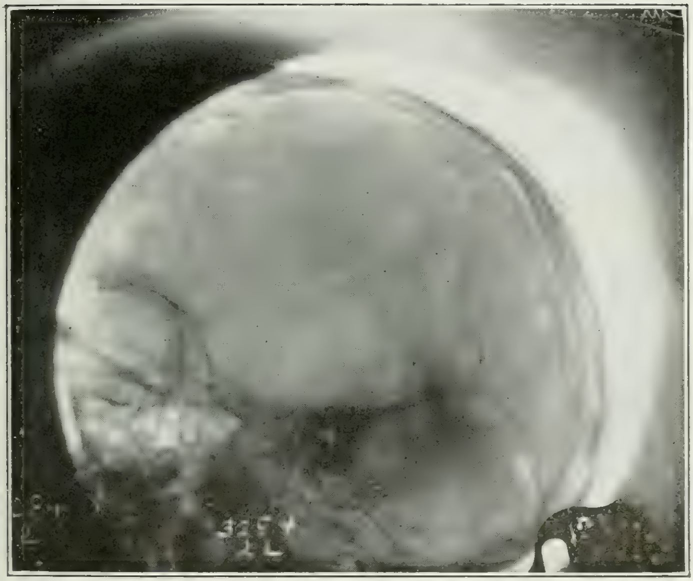
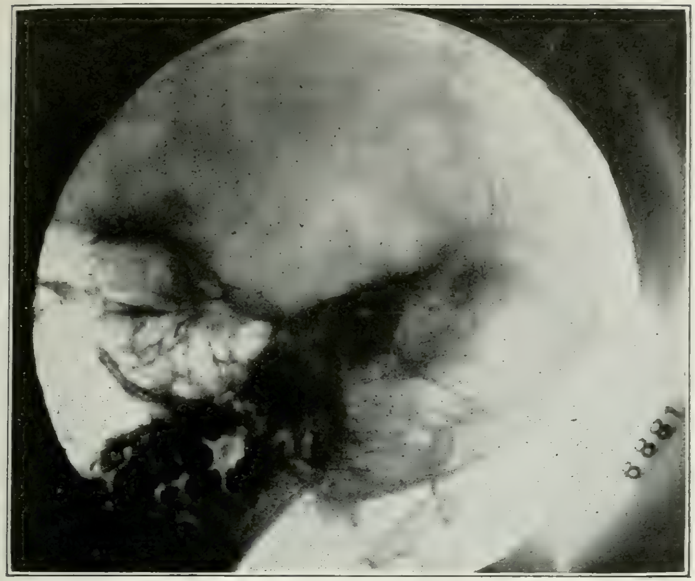


Plate 7. Figure 18.



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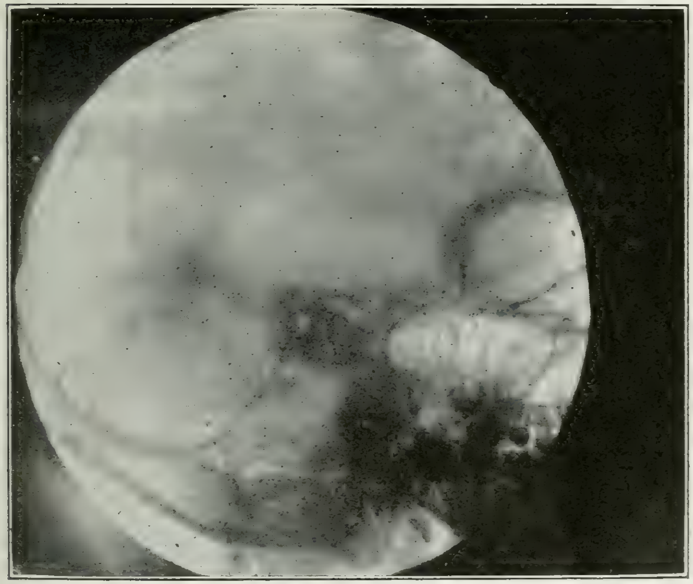

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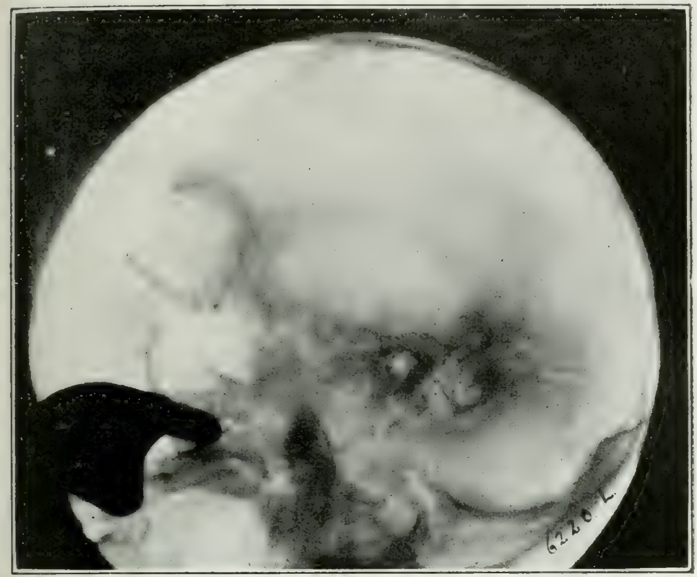

Plate 10. Figure 21.

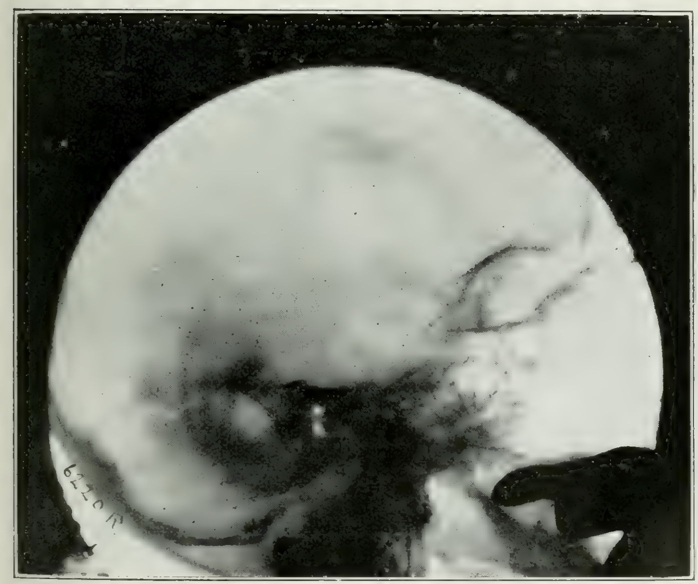

Plate 11. Figure 22.

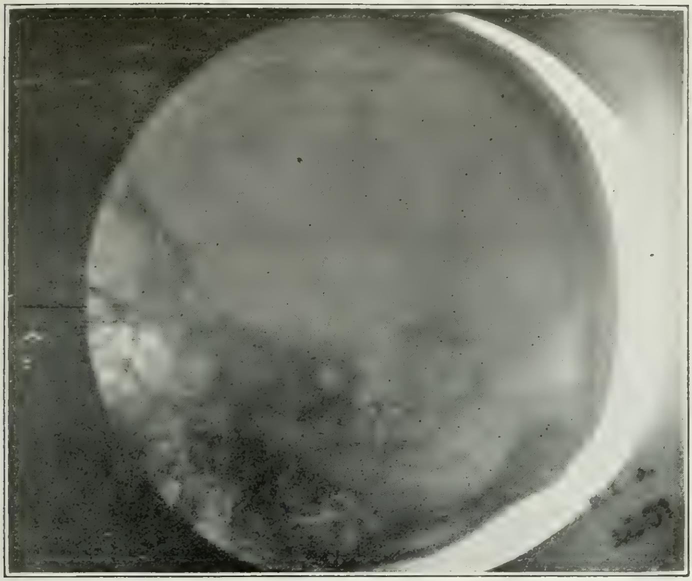

Plate 12. Figure 23.

Plate 13. Figure 24.

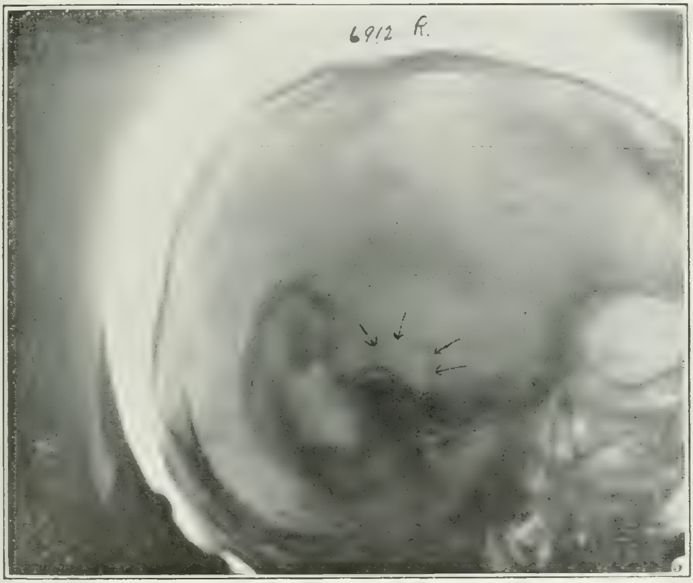

Plate 14. Figure 25.

XLVI.

SPHENOETHMOID SINUSITIS.

By John J. Shea, M. A., M. D.,

Memphis.

Wright,[1] in his History of Rhinology and Laryngology, relates of a slab which was erected in honor of the private physician of King Sahura, who ruled the Egyptians in 3500 B. C. From the translations it appears the physician cured a nasal infection of the king, so the king honored the ancient rhinologist and his wife by having the royal stone cutter inscribe the deed, so future generations might appreciate that the nares of even an Egyptian king could be pathologic, and that rhinologists of his day had adequate knowledge and were sufficiently versed in their art to cure the malady.

From ancient time until the present, acute and chronic infections have affected the upper respiratory tract. Formerly they were called rhinitis, and we were taught the symptoms and treatments of hypertrophic rhinitis, and of atrophic rhinitis, and of rhinorrhea, but today we appreciate that the membrane of the nares is the show window for the sinuses, and every rhinologic examination has as its fundamental basis a study of the accessory sinuses. The recent epidemic of influenza was thought by many to have been primarily a sinus infection, and subsequently a septicemia. Whether this deduction is correct or not, we are seeing five cases of sinus infection now, where we saw one of the disease prior to the epidemic.

Not all of the cases give a concise history of having begun with the "flu," but it is nearly universal for them to relate the influence the infection had upon their disease. The disease is found in three stages: acute, subacute, and chronic. The acute stage can be treated medically, and the subacute will often subside under medical treatment, but the chronic stage is seldom more than improved by anything less than an exenteration of the ethmoids and a drainage of the sphenoid.

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*Candidates' Thesis for the American Laryngological, Rhinological and Otological Society, accepted January, 1922.

The use of radium for the relief of the postoperative headache when it is severe, and its application for the removal of the excessive granulations and recurring polyps is worthy of presentation. The ethmoids may act as a focus of infection and either directly or indirectly cause an inflammation of the optic or auditory nerve or both. The chief complaint of these patients is not always nasal, but may range from progressive loss of vision or hearing to a persistent cough with a loss of appetite and fear of tuberculosis. Mental symptoms are very common.

ANATOMY.

The ethmoid and sphenoid sinuses are so closely articulated as to be considered one structure. The line of demarcation between the anterior and the posterior ethmoid cells cannot be made out surgically, and in a great number of noses the relationship of the posterior ethmoid cells and the sphenoid is so complex that a complete exenteration of the ethmoids will require the removal of pars nasalis and the pars ethmoidalis.

The ethmoidosphenoid sinuses constitute a series of cells of varying communications formed by the ethmoid, the frontal and sphenoid bones, and limited by the superior maxillary, lacrimal and palate bones. The middle turbinate and the rudimentary superior turbinate are a part of the ethmoid bone.

The relationship of the sphenoid to the posterior ethmoid cells is not constant. In the majority of cases the sphenoid articulates anterolaterally with the posterior ethmoid cells, but there are many instances where the cells will surround the sphenoid superiorly.

The size of the middle turbinate is often dependent upon the condition of the ethmoids, and in those cases where the bulla has hypertrophied to the cystic state the middle turbinate will be displaced and moulded between it and the septum, and at times atrophy. The posterior end of the middle turbinate bears a constant relationship to the sphenoid sinus and can be relied upon as a landmark for the puncturing of the sinus.

The sphenopalatine and the anterior and posterior ethmoid arteries supply the ethmoid and the sphenoid, and the location of the sphenopalatine artery should be kept constantly in mind while working in the sphenoid region, for injury to it will promote free hemorrhage and handicap the procedures.

The sphenopalatine artery is the terminal branch of the internal maxillary, and enters the nasal fossæ through the sphenopalatine foramen. The sphenopalatine foramen is formed by the articulation of the palate bone and the under surface of the body of the sphenoid, and transmits the sphenopalatine artery and the nasopalatine and superior nasal nerves.

The nerve supply is from Meckel's ganglion and the nasal branch of the ophthalmic; therefore block anesthesia is applicable in this field.

Mullins,[2] of Colorado Springs, worked out the lymphatic supply of the ethmoids as follows: "The lymphatic drainage is by way of the submaxillary and deep cervical and the retropharyngeal glands to the cervical lymph ducts, then into the great veins to the right side of the heart, and the pulmonary artery conveys the drainage to the lungs."

There is a type of ethmoids which may be termed "persistent infantile," inasmuch as it represents the undeveloped variety. The cells are small and few, with thick walls. The frontal sinuses are small or rudimentary, but occasionally there is a supraorbital ethmoid cell larger than the frontal, but, as a rule, in this type there are no atypical cells.

The morphology of the ethmoidosphenoidal cells varies to such an extent that we find cells which extend across the orbit for the full width of the frontal sinus, while at other times they encircle the sphenoid sinus superiorly or extend down into the pterygoid process. Similar to the mastoid process, there are times when it would be impossible to exenterate all the cells.

The sphenoid sinus occupies the body of the sphenoid bone and has six walls:

1. The anterior wall or face of the sphenoid, which is divided into the pars nasalis, in which is the ostium and the pars ethmoidalis, which articulates with the ethmoid bone completing the posterior cells.

2. The inferior wall, which forms the vault of the nasopharynx.

3. The posterior wall, which is the thickest and which is composed of the cancellous bone of the sphenoid.

4. The superior wall, which is the floor of the sella turcica. This wall is in direct contact with the optic nerves and optic chiasm. The dura mater reduplicates itself and stretches over the sella in such a manner as to make this space extradural. Hence, accidental puncture through the superior wall is not lethal, due to its being extradural.

5. The external wall, which is the thinnest and is in direct contact with the cavernous sinus. (Case 3.)

6. The septal wall, which may be deviated. The two sinuses are seldom of equal size. The cavity is lined by a very thin membrane which strips readily.

ETIOLOGY.

Age.—Dean[3] has called attention to the number of ethmoid and sphenoid cases found in children. No age appears to be exempt, each favoring the variety corresponding to its activities —that is, in children the usual inflammatory diseases are acute, so in sinusitis we are more apt to find an acute than a chronic sinusitis in children. In children we see the acute or subacute with active symptoms. Dean[3] reports a case found at autopsy in a child sixteen months old. The sphenoid, though present at birth, is less of an anatomic entity than the ethmoids, but after the fifth year it assumes its proper importance. In the young adult we see the above and a few of the atrophic varieties, while in the adult and the aged we see the acute, the hyperplastic and the chronic suppurative with its polyps and purulent discharge. The cases in the children act as foci of infection, while in the adult their effect is mostly direct.

Sex.—Males are slightly in the majority, due to their greater exposure and less care during the acute stages.

Social Status.—In the clinics the cases appear to be more severe than those met with in private practice, but this may be due to the fact that the clinic cases wait longer before they consult the surgeon.

Bacteriologic.—In turn, all of the pathogenic bacteria have been accused of being the cause of acute infection of the upper respiratory tract, and now Foster[4] claims a filterable virus to be the exciting cause of the acute cases. This confusion of knowledge is the reason for the failure of vaccines receiving a better welcome. But whatever was the active organism of the late influenza epidemic, it seemed to be very potent in activating an infection in the sinuses. Cultures from the acute cases vary from day to day, and those obtained in the chronic cases may be from a secondary infection.

Protein Sensitization.—Protein sensitization has also attracted attention because of its influence in these cases. By constant stagnation of the circulation the reparative processes are retarded and metabolic changes occur in the parts, which promote chronicity.

Internal Secretions.—Internal secretions act similar to protein sensitization by their action on the circulation of the lining membrane of the nares and sinuses.

Trauma.—In the service at Base Hospital No. 57, only one case of bullet wound of the ethmoid was seen. This was a negro private who had been hit by a machine gun bullet, which passed through both nares and a secondary suppurative ethmoiditis developed. There have been cases of accidental puncture of the ethmoids, but the percentage is very small.

Extension.—An empyema of the maxillary antrum may ascend into the ethmoid and develop an acute ethmoiditis, though the reverse of this is usually the case. Similarly the sphenoid sinus may be infected from the ethmoids.

Diseases.—Influenza appears to have a predilection for infecting the ethmoid and the sphenoid, and the present increase of the number of sinus diseases may be attributable to the recent epidemics. Erysipelas can be the result of a chronic ethmoid infection and not the cause as formerly thought. (Case 2.) The acute exanthemata are complicated by infections of the sinuses and are the chief causes of the disease in children. Syphilis, tuberculosis and malignancy may either directly or by extension cause an infection of the sinuses. (Case 18, 19, 20.)

Climate.—Climate influences the course of the infection. The worst climate is a cold, wet, heavy atmosphere with rapid changes, and the best climate is one in which there is plenty of sunshine, but with enough rainfall to keep vegetation fresh and the atmosphere bracing. The extreme tropical temperature promotes atrophic degeneration. (Cases 4 and 5.) A change of climate will stimulate the nasal circulation, and the cases acquired on the coast will improve in the mountains, while those contracted in the mountains will improve at the seashore. Sunshine will stimulate the healing during convalescence, and the acute cases are always worse after the sun goes down.

Hypertrophy of the Tonsils and Adenoids.—The hypertrophied adenoid and tonsil affect the ethmoids and sphenoid in three ways: (1) Mechanically, by blocking the discharge from the sinuses and causing stagnation, and in turn the collected discharge irritates the adenoid and keeps it in a constant state of inflammation. (2) There is a lymphatic connection which serves to spread an infection from the lymphoid tissue of the Waldeyer ring to the sinuses; and (3) by direct extension of the inflammation.

PATHOLOGY.

The ethmoid and sphenoid sinuses, being lined by mucous membrane, are subject to all of the forms of inflammation that mucous membrane present, and because of the membrane's construction, blending as it does with the periosteum, undergoes several peculiar pathologic changes.

In the acute cases there is an active inflammatory reaction of the membrane with a free flow of discharge. At first the discharge is an excess of the normal secretion, but as the disease advances, more pus cells are thrown out and the character of the discharge changes to mucopurulent or purulent. The congestion and discharge soon obstruct the ventilation and drainage of the cells, and absorption of the poison is manifested by constitutional symptoms, as fever and malaise. Under treatment the congestion is relieved and ventilation and drainage restored with a lessening of the purulent character of the discharge and of the headache. But if the infection is allowed to continue, the end result will depend upon the virulence of the infecting organism. In mild cases an immunity is soon developed and the inflammation subsides. This may be completely or with a remaining focus subject to reinfection. The severe cases completely obstruct the drainage, and the infection passes through the bony wall, causing a periostitis of the ethmoid orbital wall. If a perforation results, orbital cellulitis with its train of symptoms will follow. The frontal sinus may become blocked and infected or the antrum utilized as a reservoir and an empyema of the maxillary sinus complicate the situation.

From infancy to the grave the membranes of the sinuses are constantly being abused, and the chronic conditions are brought about by the summation of the acute infections. In the cases where a focus of infection remains, the mucoperiosteal membrane is constantly congested, which interferes with growth and repair, and in time the bony walls of the cells become like paper and the ethmoids undergo cystic changes. There is an influence, the exact nature of which is not thoroughly understood, exerted by the endocrinal secretions. Theorizing, it may be that during the systemic stage the toxin influences the internal secretions and they in turn are deficient during the reparative stage of the inflammation, and in this way the atrophic condition of the bony walls develops.

In persistent infantile ethmoids the inflammation is not so active, but is more painful and more likely to become chronic. The normal pneumatic structure of the ethmoids allows a greater extension of the infection, with less probability of complication and chronicity, than the sclerotic ethmoid with its thick walls and small cells.

Extension of the infection to the bone will cause an acute osteomyelitis. This is more common in children than in adults and more common in the acute stage than in the chronic stage.

Briefly, the stages of pathology of the ethmoids are: (a) Acute, either catarrhal or purulent; (b) chronic, usually purulent; (c) atrophic and cystic; and (d) hyperplastic. The difference in the last two appears to be due to the character of the reparative process assumed by nature in its attempt to eradicate the disease.

The sphenoid sinus differs in its behavior towards infection, due to the fact that it occupies the center of a more massive bone, and is more abundantly supplied with nutrition. The acute and chronic suppurative changes occur after the same fashion, but cystic changes are never seen, though the thinness of the anterior wall at times is presumption that it too undergoes paperization. Where there is a marked difference in the size of the two sphenoid cavities and they both are infected, the smaller of the two will require a longer postoperative period to heal or will more readily become chronic. The anterior wall of the sphenoid springs from cancellous bone and is prone to produce excessive granulations at its base, which will close in any surgical opening unless care is taken to retard it and allow the epithelium to heal over the edge of the wound.

Whether polyps spring from the superficial layer and by absorption cause the underlying bony changes, or whether the bony change occurs first and the polyps result therefrom, is a mooted question, but today the opinion is that the polyps begin in the mucosa. Nevertheless as long as polyps reform, there is present pathology in the bone, and removal of their base will be necessary for a cure.

The lymphoid tissue of the pharynx and the larynx undergoes hypertrophy and congestion, due to the irritation from the discharge and as a part of nature's fight against the infection. The mouth of the eustachian tube may become obstructed and a eustachian salpingitis develop. An inflammatory hypertrophy of the subepithelial lymphoid tissue, constituting the lateral pharyngeal wall, and the posterior pillar of the tonsil, signifies a postnasal discharge and is convicting evidence of sinus disease.

CHRONICITY.

Each succeeding acute infection interferes with the nutrition of the membrane of the sinus and its bony framework, and through faulty metabolism the process of repair is weakened. The cells may become cystic or the membrane undergo polypoid degeneration. For the tissue to atrophy the destruction must be greater than the repair, and there must be a lessening of circulation. Here the role of the endocrins is brought into play. The persistent infantile type of ethmoid will, like its prototype, the sclerotic mastoid, readily become chronic. The pneumatic nature of the ethmoids permits of severe infections, with vicious inflammation, without loss of tissue.

CLINICAL HISTORY.

Chief Complaint.—The patient seldom gives as the chief complaint a symptom which is a direct accusation of the ethmoidosphenoidal sinuses. Because the discharge descends from the posterior nares into the nasopharynx and down to the larynx, the patient's attention is centered upon these regions, and he complains of a clearing of the throat and an irritable pharynx or a productive cough, with or without hoarseness. If there is a marked hypertrophy of the ethmoids or the turbinates, blockage of nasal ventilation will be the principal symptom. White[5], of Boston, has attracted our attention to seeking a cause in the ethmoid for progressive loss of vision.

Likewise, we are constantly meeting cases of progressive deafness, which have as their cause an infection of the ethmoid or sphenoid sinuses, and whether the aural condition is due to extension of the infection through the tube or by the action of the toxins on the eighth cranial nerve is difficult to decide. (Case 29.)

When the sinuses are acting as foci of infection, the patient may complain of many and varied symptoms. This is best exemplified by the arthritis of childhood, as admirably pointed out by Dean[3] and his colleagues.

The west is full of patients who have been urged to seek a better climate before their pulmonary condition advanced beyond repair. They had sought relief from a cough that was productive upon arising in the morning, loss of ambition, increasing lassitude, and an afternoon elevation of temperature of a degree or so. This train of symptoms attracted the attention of the diagnostician towards their chests, and he, failing to find any physical signs, had their lungs radiographed, with the usual findings, "peribronchial thickening, mediastinal cloudiness, and a few apical scars." During all this time the case is still having a postnasal discharge, and each morning finds the discharge about the larynx ready to be expelled. Hence the diagnostician, in despair, advises "Go west," instead of sending them to a competent rhinologist. (Cases 6, 7, 8, 9, and 10.)

Headaches.—The typical headache of sphenoid sinusitis is in the suboccipital region or deep in the center of the head, radiating to the surface at the vertex. The anterior ethmoids will cause a headache through the temples and over the eyes, with pain on using the adductor muscles of the eye. The posterior ethmoids, by their influence on Meckel's ganglion, may cause pain in the various distributions of the ganglion, but the most notable point being two inches posterior to the external auditory canal (Sluder[6]). There is a depression of cerebration in these cases and the memory suffers. Concentration is difficult, but all can be cleared away by removal of the cause. (Cases 2, 3, and 28.)

PROGRESS.

During the acute period, discharge and interference with nasal ventilation constitute the chief complaints of the patient, while during the subacute stage the obstruction will be greater or less, depending upon whether the cells hypertrophy or atrophy. The discharge at first remains mucoid in character, but greater in amount, becoming mucopurulent and finally purulent. If the acute infection is caused by the hemolytic streptococcus, the discharge will be tinged with blood. (Case 1.) The subacute stage may last throughout a winter and with spring the symptoms and signs decrease, completely to a cure or incompletely to the chronic stage. The lack of reaction marks the chronic stage, and the progress becomes retrogressive without inflammatory reaction, except whenever there is an acute exacerbation. With each of these acute exacerbations the chronic condition becomes worse, until the atrophic or cystic stage is reached. It is difficult to express a prognosis on whether or not the tissues are capable of regeneration. When least expected the tissues make a favorable postoperative recovery, and in the end healthy membrane may be seen where before were scabs and an atrophic membrane.

DIAGNOSIS.

The diagnosis of ethmoidosphenoid sinusitis required a thorough examination of the nares, transillumination, suction and a legible radiogram.

Inspection.—Occasionally there will be a dermatitis about the vestibule of the nose from the irritation of the discharge. especially in children. The condition of the membrane of the septum and the turbinates will range from an active congestion to a boggy edematous infiltration. During the acute stage all erectile tissues will be congested, while in a number of chronic cases the membrane will demonstrate that there is a deficiency of vasomotor control. Discharge may be seen coming from under the middle turbinate or posteriorly in the choana above the inferior turbinate. If this is wiped away and shortly returns in the same region, then one can feel certain that it is not being secreted by the exposed membrane, but is coming from the sinuses. Polyps generally denote sinus infection, and their position is a help in determining which sinus is diseased. Hypertrophy and congestion of the subepithelial lymphoid tissue of the pharynx are convincing evidence of a postnasal discharge, and the presence of the discharge in the nasopharynx is a verdict against the sinuses.

Transillumination.—Transillumination is better for studying the maxillary and the frontal than the ethmoid sinuses, but when these cavities illuminate clearly and the nares are darker than normal one may suspect the ethmoid cells of being filled with discharge or their membrane thickened by chronic inflammation.

Radiography.—To Law, of the Manhattan Eye and Ear Hospital, is due credit for perfecting a technic that will give a surgical picture of the sinuses. Following Law's technic, the ethmoid cells will be thrown free from the other shadows and will stand out in bold relief. The anteroposterior picture will display the maxillary, ethmoidal, frontal and sphenoidal sinuses, while the lateral picture will show the posterior ethmoid and sphenoid sinuses and the depth of this frontal sinus. Taking the ethmoid, the frontal and the orbit, the two sides should be compared, and in unilateral involvement the contrast will be marked. If the lateral picture shows the frontal to be of average depth, the anteroposterior picture in normal sinuses will show the frontal ventilated and not darker than the ethmoid cells, or lighter than the orbit. When the frontals are cloudy the ethmoids will be cloudy or hazy. There is a common picture of the frontal being of average size, but more cloudy than its fellow, or when the two sides are concerned both frontals being cloudy, but still the ethmoids appear well ventilated. The frontals should be no more cloudy than their corresponding ethmoids, and if they are it means that the ethmoids have advanced from the inflammatory to the cystic state. At operation this variety of ethmoids will show atrophy, and in the past the radiogram reading in these cases was "ethmoids appear well ventilated." The truth of the matter is that they are too well ventilated. Careful scrutiny of the ethmoids will show cystic changes. This is best illustrated when there is a supraorbital ethmoid cell well ventilated under a cloudy frontal sinus. Discharge and thickened membrane will equally shadow the ethmoid region as shown by taking a picture before and after the use of vacuum treatment.

When the lateral picture shows the sphenoid to be of average size, it should be visible on the anteroposterior view as a heart-shaped object about half way up the nares. If its outline is blurred, a diagnosis may be made of thickened membrane or pus in the posterior cells and sphenoid sinus. Occasions will arise when the comparative measurements of the two sides will not determine whether or not the space under consideration is the sphenoid or an atypical posterior ethmoid cell, and the only positive means of deciding will be to take a lateral view with probes in the ostia of the sphenoids.

The adverse criticism of the value of the radiogram in sinus diagnosis has been due to the poor pictures rendered for interpretation. No diagnosis could be made from some of them, for the sinuses were not in surgical relationship and their contours distorted. By following the technic of Law, the details of the sinuses can be brought out in such a manner that reliable information may be obtained, and each surgeon should be able to read his own pictures and by studying the views after the operation develop a better knowledge of this most important adjunct. Formerly the only information desired by the surgeon was the size of the sinuses, but today we expect to obtain an inkling of the pathology and a guide as to the best surgical procedure.

Suction.—The use of vacuum properly applied will drain the sinuses of their discharge, and by observing its location and character valuable knowledge may be obtained. It is the constant discharge as shown by suction that convicts a sinus. If the discharge is to be studied bacteriologically the nose should be cleansed with normal saline, then suction applied to withdraw the discharge, after which a sterile glass tube should replace the nasal tip, and suck a specimen from under the middle turbinate or from the posterior naris.

TREATMENT.

Rest is the most important therapeutic agent in the acute and the subacute stages. A patient is always better after a good night's rest.

Acute Stage.—Not all suppurative inflammation of the ethmoidosphenoidal group requires operation. Many of the acute cases subside of themselves and others can be hastened by promoting better ventilation and drainage. The use of an adrenalin spray with a 1/4 per cent cocain solution as the basis will shrink the congested turbinates and the membrane lining the septum. The judicious application of suction will aid the drainage, and the removal of the discharge from the nares by a saline douche will give comfort to the patient. The patient can be furnished a water suction device of the Brawley or Gray type for home use, and also be taught how to irrigate the nares without danger to his ears. The headache of the acute cases will be relieved by ventilating the sinuses with the above treatment, and in those obstinate cases with severe headache 5 mm. of pituitrin extract (surgical) hypodermically will constrict the vessels and open the way to ventilation. If this plan is followed during the first day of the socalled influenza cases there is less absorption of toxins and the case will assume the characteristics of the "three days' fever" malady. An oil spray of guaiacol will serve well as an emollient to the washed membrane, and also act as an antiphlegmatic.

Subacute Stages.—Before advising an operation the above treatment should be ordered for daily use at home. Sustaining treatment and the administration of autogenous vaccine will aid. If there is a deflected septum or a hypertrophied turbinate, or both, the correction of these deformities will afford better ventilation and drainage. The ethmoid is so constructed as to handle infections if given an equal chance. There is a future for endocrine therapy in these cases, for some improve under thyroid gland, gr. 1, twice a day.

Chronic State.—The above treatment will often be necessary to convince the patient that he is suffering from a more persistent and pernicious ailment than a "head cold" or "hay fever." The cleaner the cells are of discharge the less risk there will be of infection at operation and of postoperative fever. If a submucous resection of the septum is needed, it had better be done as a preliminary step, and there are some surgeons who will at this time amputate the middle turbinate, but the latter structure is a guide as to the situation of the cells and its posterior attachment a landmark for the puncturing of the sphenoid sinus and had better remain until the exenteration of the cells is attempted.

Postoperative Stage.—Whether the intranasal or the radical operation is done, the postoperative care must be carefully managed and not delegated to an assistant. The restoration of the two functions of ventilation and drainage is necessary for a success. If there has been no packing the nose should be sprayed frequently with normal saline and an oil instilled to prevent the formation of scabs. Where packing is employed it should be kept moistened with saline solution and its removal effected as soon as possible, and at the end of twenty-four hours if feasible. The less handling of the parts until the granulations have become organized, the more rapid will be the improvement. When the granulations show signs of becoming excessive, steps should be taken toward their eradication. Silver nitrate or the actual cautery will destroy the hypertrophied granulations and allow the epithelium to cover over the exposed surface. If the granulations become very excessive or small polyps reform in spite of treatment the use of radium is advised.

When to begin the use of the suction will depend upon the discharge. If the discharge is blocking the passage and simple douching cannot remove it gentle suction may be tried.

The task of keeping the sphenoid sinus open is often difficult. The epithelium must cover over the edges of the opening or it will decrease in size until a new anterior wall has formed, thereby blocking the sinus. If the sphenoid sinus does close, dilatation of the opening with cotton tampons or its removal with the biting forceps will be necessary to effect a permanent opening. This may be repeated many times before it becomes permanent.

Radium.—The task of keeping granulations under control is difficult. Radium will serve better than any other measure. The granulations spring from the cut surface and serve as a supporting framework for the epithelium to grow over and heal the wound. If the granulations become too excessive the epithelium cannot surmount them. The experience in Case 23 showed two things: First, that radium will reduce excessive granulations and scar tissue in the ethmoids and sphenoids, and secondly, that it will give more than temporary relief from the headache.

The radium by its action on the new formed tissue produces a fibrosis with a relief of the pressure. The nares are packed off and protected, except for the part to be treated, and the needles encased in wax and placed in apposition to the ethmoids and retained there by cotton plugs. The first dose is usually for one hour of 50 mg., and depending upon the reaction, the second dose is either increased or diminished or more thoroughly screened. Within two weeks there is marked improvement. We have asked of radium the impossible. and because it cannot perform a miracle we are disappointed, but its use in the removing of excessive granulations and polypoid degeneration of these wounds is possible and can be relied upon. The idea has been advanced that the radium tends to produce a fibrosis, and when this tissue is removed it will not return, whereas when the granulations are removed their recurrence is nearly certain. This is shown by treating a very pernicious reformation case with radium and then removing the excessive tissue.

OPERATION.

The question of the choice between the intranasal exenteration of the cells and the external radical operation will depend upon:

(a) The size of the sinuses; the larger sinuses with ramifications will demand the radical operation.

(b) The cases complicated by a chronic infection of the frontal sinus will be better treated by the radical method.

(c) Those cases in which the ethmoid cells are acting as a foci of infection will require a thorough exenteration as only the radical procedure affords.

(d) If the intranasal operation has been thoroughly done and the after treatment carefully managed, with no improvement in the symptoms, the radical is indicated.

The technic of the Lynch frontoethmoid operation is the best for the radical ethmoidosphenoidal procedure.

The best operative position for the patient is half reclining on a full length operating table.

Thorough anesthesia of the parts may be obtained by swabbing the surface of the turbinates, the ethmoids and sphenoids with a 4 per cent solution of cocain to which ten minims of adrenalin solution to the ounce has been added, and then injecting the region of Meckel's ganglion with a 2 per cent solution of novocain. The postnasal branch of the sphenopalatine may be injected on the anterior surface of the sphenoid, and a similar injection at the anterior attachment of the middle turbinate will anesthetize this region. By its action on the sympathetic element of the ganglion vasor constriction of the area is produced. In cases where there are many polyps and the ethmoids are markedly congested, the above will not control excessive bleeding, but if pituitrin be given intramuscularly throughout the operation, the field will be less sanguinous. Only two or three minims every fifteen minutes will be sufficient and will greatly expedite matters. Half an hour before the operation a hypnotic is administered, and if the necessity arises subsequent doses are given.

Before starting the operation a small piece of gauze is inserted deep into the nares against the inferior wall of the sphenoid sinuses. This prevents blood from dripping down into the throat, and thereby avoids the delay occasioned by the patient constantly clearing his throat.

The middle turbinate is thoroughly amputated by cutting its membranous attachment with a long nasal knife, and severing the bony attachment with turbinate scissors, either curved or straight, as the necessity demands. Then its removal can be accomplished with a nasal snare. There will remain a tag of varying size at the posterior attachment of the middle turbinate. Using this as a guide, the sphenoid sinus is entered by direct puncture with a sphenoid punch forceps. If the head is in a half reclining position the instrument is directed between the tag and the septum on a direct line. The sphenoid sinus will always be at a greater depth than the tag, and in this manner the tag can be utilized as a landmark in measuring. No attempt is made to locate and enter the ostium of the sphenoid. The beginner expects the sphenoid sinus to occupy a higher plane and is often tempted to puncture too high. After the first bite the resection of the pars nasalis is accomplished and then the pars ethmoidalis removed from above downwards, care being taken to respect the sphenopalatine artery. The work on the sphenoid should be done first, as it is the most delicate, and a clean field will allow of a greater precision. Next should be the removal of the posterior ethmoid cells, which articulate with the sphenoid. If there are any cells which extend above or externally to the sphenoid they should be cleaned out. All of the older methods worked from before backwards, and by the time the posterior ethmoid cells and the sphenoid were reached the operative field was so obscured as to detract from the exactness necessary for the success of the operation. Care can be taken not to wound the sphenopalatine artery, which lies in front of the external inferior angle of the anterior wall of the sinus. With a sharp biting forceps the ethmoid cells are removed from behind forward, starting on a plane with the severed attachment of the middle turbinate and working from behind forward and from above downward. The cells above the middle turbinate must be cared for properly with a small sharp biting forceps and not curetted. No tearing should occur; each grasp should acquire only what it is capable of removing by a clean bite. The anterior cells require a sharp angled forceps and at times a curette. The bulla and the membrane covering the uncinate process should be reduced thoroughly, or else the drainage of the anterior cells will be blocked during the convalescence. The cells are removed by sight and not by touch, leaving the membrane lining the remains of the cell walls, which are made up by the orbital plate. This is done with the hope of less granulations springing up from denuded bone. The cavity is flushed with ice cold normal saline solution, which will stop the capillary oozing and allow a thorough inspection of the field. Any remaining diseased tissue is removed and the frontal duct probed as to its patency, but no attempt is made to curette the lining membrane of the duct. If the hypertrophied cells about the duct are reduced the duct will drain and no stricture of it will occur from granulations, whereas, if an attempt is made to curette the duct granulating tissue will soon block the drainage of the frontal sinus.

Whether to pack or not depends on how dry the parts are at the finish of the operation. If there is free bleeding from the region of the sphenopalatine artery and crushing of the parts with an artery forceps fails to stop it, not to pack is a risk. When packing is used, gauze soaked in liquid petrolatum to which has been added tincture of benzoin compound is preferable. The distal end is anchored in the sphenoid cavity and allowed to drop down to the soft palate and out along the floor of the nose to the vestibule, where it is fixed by the assistant, and into this sack the remainder of the packing is inserted. The packing should not be allowed to dry, but should be kept moistened with either more of the oil or normal saline solution. This will aid its removal. The disadvantage of packing is the possibility of damming up infection and of hemorrhage upon its removal. The earlier that the packing can be removed without fresh hemorrhage the better it will be for the patient and the more rapid will be the healing. If secondary hemorrhage occurs on the fourth or fifth day a postnasal pack will be more efficacious and less dangerous than the simple anterior pack.

The use of suction during operation is as valuable as in throat work. At first the author used an ordinary glass drinking tube, attached to the suction part of the ether suction apparatus, but now he uses a specially constructed metal tube, capable of being boiled. Suction will expedite matters by preventing the loss of time utilized in sponging or by the patient clearing his throat.

RADICAL OPERATION.

The incision of the Lynch operation is of value because of the exposure it affords. The frontal sinus is attacked and obliterated and the ethmoid cells removed by sight. The sphenoid is readily exposed and its anterior wall removed, and if it contains polyps the lining membrane is removed, but if it is lined by a thin clean membrane this is left. The complete removal of the cells will eliminate the ethmoids as a focus of infection.

COMPLICATIONS.

The complications are of the disease and of the operative treatment. Those of the disease are due to extension of the infection and depend upon the route of the extension. If the infection perforates into the orbit there will occur an acute orbital cellulitis with its possibility. The eye may be involved from the sinus infection in the following manner: ulcer of the cornea, phlyctenular conjunctivitis, uveitis, retrobulbar neuritis, chorioretinitis, panophthalmitis and orbital abscess. An acute osteomyelitis is more common in children and acute inflammation than in adults and chronic inflammation. Mullins[2] has called attention to the number of pulmonary conditions due to ethmoid and sphenoid infection, and in children we see an inflammation of the lymphoid tissue of the pharynx and of the membrane of the larynx. Dean[3] illustrates with case histories the relationship between multiple arthritis in children and paranasal sinus disease.

Meningitis or extradural abscess is possible, but seldom seen. A general septicemia (Case 1) is more common than usually realized, and many of the fatalities in the influenza epidemics were of this type. The surgical complications are as follows:

Anesthetic.—The committee upon local anesthesia of the American Medical Association has awakened a true appreciation of the dangers of this form of anesthesia. The administration of the preliminary hypnotic reduces the dosage of cocain or its allied drugs, and also prepares the nervous system for the ordeal of the operation. Hurd,[7] in his paper on scopolamin at the Boston meeting of the A. M. A., 1921, endeavored to prove that shock entered into the socalled cocain poisoning and that the administration of a preliminary hypnotic reduced the possibility of shock. This was refuted by Mayer on the grounds that of the cases reported the symptoms pointed to poisoning and not to shock. Care must be taken in the use of cocain or its allied product, and each case is a rule unto itself. If poisoning does occur, the administration of ether is the most efficacious remedy. Sustain the heart, as the depression is most acute upon the cardiac center. By block anesthesia less drug is necessary and the risk is reduced. Accidents have been reported due to cocainization of the larynx from the too free use of cocain in the posterior nares, thereby abolishing the laryngeal reflexes and allowing obstructing clot to enter the glottis.

Hemorrhages.—Primary hemorrhage may occur from the anterior ethmoid artery or the nasopalatine. The former may be stopped with bone wax, and Yankauer[8] has advised crushing the latter with a long artery forceps at its entrance into the nasal fossæ. If the bleeding is moderate a little patience will often allow the formation of a clot. When a light pack will not control the bleeding a postnasal tampon should be used.

The use of an adrenalin spray every fifteen minutes for the first three hours and every half hour for the remainder of the first half day will aid in the prevention of late primary hemorrhage. After the oozing has increased to a flow and the nares and nasopharynx have been filled with an unorganized clot, the spray will be of no value and packing will be necessary. Ice compresses of gauze packs constantly applied will supplement the adrenalin spray.

Secondary hemorrhages are most frequent on the fifth day and may occur as late as the tenth day. If simple remedies, such as ice compresses, irrigating the nares with ice cold normal solution or adrenalin spray do not control the bleeding, the administration of morphin, gr. 1/4, and 5 cc. of hemostàtic serum should be tried. The flow down the pharynx is the indicator as to the severity of the hemorrhage and should be closely watched. If packing is to be resorted to, the postnasal tampon is the most efficacious, and will cause less absorption of toxins. Infection with a resulting slough causes secondary hemorrhages by opening greater vessels.

Surgical Accidents.—If all the work is done by sight and not by touch, and each bite removed cleanly, there will be fewer surgical mistakes. The beginner expects the sphenoid to be situated too high, and persistent attempts at entrance of the cavity superiorly will perforate the floor of the frontal fossæ. The puncture of the posterior wall of the sphenoid is difficult but possible. If the sella turcica is perforated the danger of infection is the only serious worry, and the formation of its dural canopy minimizes this possibility. If there is an escape of cerebrospinal fluid the puncture will be irrigated and infection is less probable, and when there has been sufficient loss of fluid the pressure will be reduced and the wound will close. Perforation of the orbital wall is more apt to occur when an attempt to puncture through into the antrum is tried. Of course the removal of a cell whose wall is formed by the lamina papyracea exposed the orbital cavity.

Infection.—The less packing inserted the less likelihood of infection, and of all the complications infection is the most to be feared. The majority of intracranial complications are due to the spreading of the infection by the lymphatic and the veins. As above stated, secondary hemorrhages are from infection and in a measure tend to lessen the infection. Careless puncturing through into the cranial cavity will carry along contamination and produce meningitis or abscesses.

Meningitis is as a rule acute and fatal, but extradural abscesses may be subacute or chronic, and under proper treatment drain and effect a cure. There are times when the headache increases, the temperature rises a degree or so, and the neck muscles tighten sufficiently to prevent the patient placing his chin upon his chest without suppuration developing within the cranium. These cases are irritation of the meninges and require immediate attention. If they are packed, removal of the packing is imperative, and if they are not packed, shrinkage of the stuffy membrane by the use of the adrenalin spray or pituitrin hypodermically will promote ventilation and drainage. In twenty-four or thirty-six hours the headache should improve, and by the second day the rigidity of the neck muscles should be less. If there are no improvements by this time the case should be studied from a neurologic standpoint, to ascertain if the infection has spread inwards to the meninges.

Excessive Granulations and Reformation of Polypoid Tissues.—The rapid development of obstruction to the ventilation and drainage of the sinuses by the reformation of polypoid tissue or excessive granulations does not always denote necrotic tissue. This is proven by the removal of this new formed tissue with radium. If there was necrotic bone the simple application of radium would not obliterate it. The excessive granulations are formed because the epithelium cannot grow inward from the edge as fast as the granulations can spring from the bone. The presence of excessive granulations or polypoid tissue will produce a return of the discharge and in time a recurrence of the symptoms.

Etiology.—Case 1 illustrates that ethmoid sinusitis could be the beginning of an influenza infection. Case 2 illustrates that erysipelas may have its focus in the ethmoids.

Anatomy.—Case 3 demonstrated how close the internal carotid can be to the lateral walls of the sphenoid.

Climate.—Case 4 illustrates that the tropics tends to produce atrophic changes. Case 5, same as 4.

Pulmonary.—Cases 6, 7, 8, 9 and 10 illustrate that the chief complaints may be pulmonary symptoms.

Extension from the Antrum.—Cases 11 and 12 illustrate that the ethmoids can be involved by the extensions of the infection from the maxillary sinus.

Secondary Hemorrhage.—Cases 13 and 27 illustrate secondary hemorrhages.

Methods of Locating the Sphenoid Sinus.—Cases 14 and 15 illustrate the value of the X-ray in deciding the position of the sphenoid when there is doubt as to its location.

Ocular Manifestation.—Cases 13 and 16 illustrate the influence the ethmoids may have on the vision and the rapid improvement which follows their exenteration. Case 17 illustrates how much pathology may be in the ethmoids, as in this case, to even cause an exophthalmus without any effect upon the vision.

Neoplasms and Syphilis.—Case 18 illustrates a suspected malignancy, but the laboratory report of chronic inflammation was sustained by the subsequent history. Case 19 illustrates a malignancy of the ethmoids and maxillary antrum. Case 20 illustrates a gumma of the ethmoids.

Children.—Cases 10, 21 and 22 illustrate the condition as seen in children.

Radium.—Cases 23, 24, 25 and 26 illustrate the value of radium in the postoperative treatment where excessive granulations are a complication.

Mental Symptoms.—Cases 27 and 28 illustrate the inability of concentration as seen in the sphenoid infections.

Auditory.—Cases 29 and 30 illustrate the influence the ethmoids have on aural pathology.

Complication of Meningitis.—Case 32 shows an apparent hemolytic infection of the meninges.

CASE HISTORIES.

Case 1.—Sergt. B., admitted to Base Hospital No. 57 for an acute infection of upper respiratory tract. After being up and about for two days, developed an acute left frontal headache, and by evening his temperature had risen to 102°. The following morning there was a beginning orbital inflammation with tenderness on pressure over the left frontal sinus. A septic temperature persisted in spite of intranasal drainage. On the fourth day a severe epistaxis from the left nares, which was followed by others, and in another twenty-four hours patient had vomited blood and coughed up fresh frothing blood. A blood culture was positive in the next twenty-four hours for hemolytic streptococci. A transfusion on the fifth day gave a new lease on life, and the temperature remained below 101° for the next day, but the improvement was only temporary, and the hemorrhage reappeared from all the mucous membranes. A second transfusion was of no avail and the patient died. At autopsy there was evidence of hemorrhage from the left ethmoid and a clot filled the frontoethmoidal region. Evidently the left ethmoid had been the site of the original hemolytic streptococcus infection. Free blood was found in the lungs, the stomach, the pericardial and pleural sacs, and under the capsules of the spleen and kidneys. With all the hemorrhages the tissues were not depleted. This case resembled in many respects the fulminating type of influenza. The local manifestation of infection in the left ethmoid and frontal sinuses, with the resulting orbital cellulitis, were the only differentiating features. With less local signs the case could have been diagnosed influenza, and I think many of the pernicious cases which died in twenty-four to forty-hours were of this same type.

Case 2.—S. E. M., age 36. Chief complaints were pain in left antrum, a productive cough, loss of weight and strength. For the past three years patient had been a tubercular suspect. During an acute exacerbation of the ethmoid infection patient developed a chill, temperature of 103.6, with a sense of fullness over the nose. By afternoon the blush of erysipelas was present over the usual nasal position, which brings up the question, "Are the ethmoids the focus of recurrent erysipelas which appears without a visible wound?" Examination: Nasal membranes were thin and coated with dried discharge and the radiogram showed the ethmoids to be cystic and the left antrum to be cloudy. The ethmoids were exenterated and the sphenoid was thoroughly drained. Two openings were made into the left antrum, one below the inferior turbinate and one above, by removing the ethmoid cells about the natural opening. As the membranes granulated and healed, the discharge ceased and the cough subsided.

Case 3.—J. H. G., age 44. Chief complaints were asthma and a chronic nasal obstruction for years, for which a submucous resection of the septum was performed with amputation of the anterior half of the middle turbinates in 1911. Because of repeated acute infections his tonsils were removed. In 1919 examination showed the nasal membranes to be pale and edematous and a few polyps sprang from under the stumps of the middle turbinates. The ethmoid cells were hypertrophied and the septum still deflected high to the left. The radiogram revealed a cloudiness over the ethmoids, and the sphenoids were of average size. Operation: The right ethmoid cells were exenterated and the anterior wall of the sphenoid was resected. The left ethmoid cells were more difficult to approach, due to the deflection of the septum. Upon removal of the left sphenoethmoidal partition a pulsation was visible through the lateral sphenoid wall, and an erosion of this wall by the internal carotid was suspected but not proven. No packing was inserted. Convalescence was uneventful until the evening of the fifth day, when the patient complained of severe pains through the temples and pain upon moving the eyes. His temperature was 100 to 104 degrees and he was unable to lower his chin to his chest. Examination of the nose was negative, but a cathartic was administered and an adrenalin nasal spray ordered. The next day conditions were the same, but the seventh day saw improvement, and on the eighth day nasal ventilation was free and there were no further headaches, but the movement of the head caused a sense of restriction between the shoulders for several days.

Case 4.—Mrs. C. E. G., age 26. Chief complaints were frontal headache and a recent acute maxillary sinusitis. Ten years ago, while living in southern Louisiana, her nasal infection took on the nature of an ozena (a condition often met with in cases which go farther south, as into Central America). Examination showed the nasal membranes to be atrophic and the remains of the turbinates were bathed in a purulent discharge. Suction was productive of more purulent discharges from the ethmoid region. The radiogram revealed a cloudiness of both ethmoid regions and an increased density over the left maxillary sinus. The frontal sinuses were clear and the sphenoid sinuses of average size. Operation: The right ethmoid cells were easily worked out, though they extended high, but the left cells had an abnormally firm bony capsule. This same condition held true with regard to the sphenoids, and only after careful measuring and by direct puncture was the left sphenoid entered. A persistent oozing from a high posterior ethmoid cell complicated affairs, and the region could not be worked out as thoroughly as the right side. This was productive of profuse granulations in this region and required energetic postoperative attention before thorough drainage was established.

Case 5.—E. W., age 11. Chief complaint was a chronic nasal infection, which had the history of an acute sinus infection while in the tropics that was complicated by an atrophic rhinitis. Examination showed the turbinates to be small, and there were many adhesions between the septum and the turbinates. At operation the adhesions were dissected and the turbinates made free. Wax plates were placed between the septum and the freed turbinates and kept there until the raw surface had healed. The postoperative treatment consisted of the daily use of suction and thorough nasal irrigation. These cases of acute sinus infection will become atrophic if the patient moves into the tropics.

Case 6.—Mrs. R. D. C., age 37. Chief complaints were obstructed nasal breathing, profuse purulent discharge, both anteriorly and posteriorly, and a persistent productive cough. Examination showed the ethmoids to be hypertrophied and that the discharge was coming from under the middle turbinates. Radiogram showed all the sinuses to be cloudy and the sphenoid sinuses of average size. Operation: A bilateral exenteration of the ethmoids and sphenoidotomy. The tissues were very congested and oozing was very profuse during the operation and required packing. Convalescence was uneventful, and as the granulations healed the discharge became less and the coughing stopped. This case had been studied for years as to the cause of the coughing, but the postnasal discharge had never been considered.

Case 7.—McK., age 46 Chief complaints were obstructed nasal breathing, asthma and a persistent productive cough. Family history of one brother out West for tuberculosis. Examination showed nares filled with polyps to the very outlet. Radiogram revealed all the sinuses to be cloudy. Operation: A bilateral exenteration of the ethmoid cells and sphenoidotomy. There was free oozing during the operation and the nose was packed. The hemorrhage upon unpacking was so profuse as to require consideration of repacking. Convalescence was uneventful, and as the acute postoperative stage passed away the symptoms grew less, and inspection two months later showed the sinuses well drained and the coughing reduced to a minimum.

Case 8.—Mrs. T. W. G., age 30. Chief complaints were that in 1919 had influenza and since then a productive cough for which she had been sent to Texas as a suspected tubercular patient. She also complained of a frontal headache, worse on the left, and tenderness of the scalp over the left parietal region. Examination showed the nasal membrane to be congested and the turbinates jammed. The septum had a spur to the left. Transillumination failed to throw any light upon the case. All the sinuses illuminated clear, due to the thinness of the facial bones and skull. The radiogram showed frontals to be rudimentary and the right ethmoid sinus cloudy. The left ethmoids were cystic. Operation: A bilateral exenteration of the ethmoids and sphenoidotomy. A submucous resection of the nasal septum was necessary to obtain working space on the left side. The ethmoid cells were found to be cystic and the right cells were filled with granulations. Convalescence was uneventful, and the case made a happy recovery with a lessening of all the symptoms save for the tenderness of scalp.

Case 9.—S. D., age 68. Chief complaints were a persistent productive morning cough and a postnasal drip during the day which required constant clearing of the throat. Past history of a chronic nasal infection and bronchitis during the winter months. Examination showed the nasal membrane to be congested and the middle turbinates displaced by hypertrophied ethmoid cells. Radiogram showed all sinuses to be cloudy and the sphenoid sinuses of average size. Operation: A bilateral exenteration of the ethmoid cells and sphenoidotomy. The right sphenoid remained well opened, but there was a tendency for the left sphenoid sinus to close by reformation of the anterior wall. The cough improved, but with each change of the weather there is a return of the coughing on rising, but it produces a clear mucus and not the former purulent discharge.

Case 10.—G. G., age 12. Chief complaints were an acute daily elevation of temperature to 100° with a productive cough. Past history: Just one year ago seen with a similar condition, and as there was a reformation of the tonsils and the adenoids, a second tonsil and adenoid operation was performed. For two weeks after the operation she had a dry irritable cough, which gradually improved. One acute infection of the sinuses with a cough during the winter. Examination: Lungs showed signs of enlarged glands and the radiogram was suggestive of mediastinal pathology. There was a postnasal discharge, and the larynx was very red. Treatment by the use of suctions and the nasal douche did not materially aid, so the child was sent to Colorado for the summer. As soon as the postnasal discharge cleared the cough stopped and the chest signs disappeared.

Case 11.—Mrs. W., age 30. Chief complaint, acute suppurative maxillary sinusitis of dental origin. Duration of one year with inferior dental treatment. At operation the ethmoid cells were found to be degenerated and filled with pus, and a subsequent exenteration of the cells was necessary to cure the antral infection. The ethmoid cells of the opposite side were not involved. This is an example of involvement by extension.

Case 13.—Mrs. B. C., age 64. Chief complaints were a cough from a postnasal drip with general muscular pains, more pronounced in the posterior cervical region. Examination: Nasal membrane coated with a thin purulent discharge, and the turbinates were atrophic. Operation: A double ethmoid exenteration and sphenoidotomy with no postoperative packing. Convalescence was uneventful until the fifth day, when a secondary hemorrhage developed from the right sphenoid region, which required a postnasal plug before it was controlled. Packing removed on the eighth day, with no difficulty, though the discharge was offensive. On the tenth day another sharp hemorrhage developed, which required anterior and posterior packing. The next day the anterior packing was removed and the following day the postnasal plug was removed and following in its wake was a tablespoonful of foul discharge. No further packing was necessary and convalescence was natural. This case was the most marked secondary hemorrhage encountered and exemplified the necessity of a postnasal plug when a sharp hemorrhage is from the region of the sphenoid.

Case 14.—J. F. H. Chief complaints were coughing, postnasal discharge, and a history of an atrophic rhinitis for twenty years. Examination showed a typical atrophic rhinitis with large foul scabs and small turbinates. Extensive treatment failed to accomplish anything save temporary relief. The radiogram showed the ethmoid cells to be better ventilated than the frontals, and the anterior posterior picture showed the sphenoid sinuses to be unequally developed, the left being the larger. The frontals were small. Operation: Bilateral exenteration of ethmoids and sphenoidotomy. There was free bleeding from the right sphenopalatine artery which required packing. Several hours after operation there developed a hematoma of the right orbit from the hemorrhage, continuing and making its exit through the sphenopalatine foramen. Convalescence was uneventful save that the right sphenoid sinus was closed when seen after an interval of a month. An opening into the sinus revealed it to be very shallow, but a radiogram was made with a probe in each sinus, and it showed that they were open. The left sinus was filled with Beck's bismuth paste, X-rayed to ascertain its size, and it was found that the left sinus was very large and that the right sinus was small, with the sphenoid partition deviating to the right. After all the granulations had healed the membrane was moist and healthy and there were no more scabs. The irritation of the larynx is less and there is no further coughing except when exposed to an irritating atmosphere.

Case 15.—W. F. M., age 24. Seen in consultation to determine if the left sphenoid sinus was open. There had been a curettement of the left ethmoid cells and what was thought to be the left sphenoid had been entered. The opening was situated high and posteriorly, but too far externally for the sphenoid sinus. An attempt was made to puncture into the sphenoid sinus, but the anterior wall was resistent and what appeared to be the anterior wall measured 8.5 cm. from the tip of the nose. A probe was placed in the cells that was suspected of being the sphenoid sinus and X-rayed and, as the picture showed, it was an ethmoid cell situated above and external to the sphenoid, and an entrance into the cranial fossæ could have been easily accomplished by the use of undue force or persistency.

Case 16.—W. A. L. Chief complaint was a progressive loss of vision to 1/200 in left eye, with central blind spot. Narrowed blurred field. Fig. 1. Acute pain about left eye for the past three weeks. Physical examination was negative, Wassermann negative. The ethmoid cells of the left side were hypertrophied, displacing and jamming middle turbinate against septum. The septum deflected to the right. X-ray revealed a large cyst of left ethmoid. Right ethmoids showed increased density. Septum to the right. Sphenoids of average size and slightly cloudy. Left middle turbinate upon removal had become flattened by atrophy. The bony septum was very thin from pressure. Upon puncturing the bulla the cyst was entered. Exenteration was simple. Anterior walls of the sphenoid removed. The ethmoids had undergone paperization and extended backwards and inwards until they completely obscured the sphenoid. No discharge present. Uneventful convalescence. Vision twelve days postoperative: Right 20/20, left 2-1/200. Four weeks postoperative, vision 20/20 both eyes. Fields and tension normal. Fig. 2. X-ray shows septum in midline and left ethmoids absent. (In order to demonstrate a cyst of the ethmoid its walls must be intact.) Case 17, Mrs. C., demonstrates the inability to show a cystic cavity after part of its wall has been removed.

Case 17.—Mrs. H. C. Chief complaints were progressive obstruction to nasal ventilation and drainage of one year's duration, with acute pain about eyes for past two weeks. There was an exophthalmus of the left eye. Examination: Nares completely filled with polyp and a profuse purulent discharge. The left eye was displaced downwards and outwards with exophthalmus of 6 mm. Vision, 20/20 in both eyes, with normal fields. The general aspect of the left orbit presented signs of subacute orbital cellulitis. Upon palpation a cystic enlargement could be made out from the floor of frontal sinus at its inner angle. No tenderness was present. Exenteration of the polyps and degenerated ethmoid cells and drainage of sphenoid sinus was done at one sitting. Convalescence was protracted due to drying of the purulent discharge with the production of scabs. X-ray six weeks after operating failed to outline the tumor mass in the left orbital frontal wall. This was due to a partial removal of the wall of the cyst. The exophthalmus persisted, but the vision remained 20/20 in both eyes before and after operation. The discharge grew less and localized to the left naris in its anterior aspect. One day while making pressure over the tumor mass a stream of the purulent discharge was visible coming down from in the region of the frontal duct. This decided the issue that our enlargement along the floor of the frontal sinus was a purulent collection and connected with the frontal sinus or a supraorbital ethmoid cell. A radical operation was advised, but as there was no further pain and the discharge was minimum she decided to wait. This case illustrates how extensive and prolonged pathology may be in an orbit without affecting the vision.

Case 18.—Mrs. F. A. P., age 54. Chief complaints were obstructed nasal breathing and profuse purulent nasal discharge. Examination showed both nares filled by polypoid tissue and same coated with a purulent discharge. Operation: The polyps were removed and the right ethmoid cells exenterated and the right sphenoid sinus drained. The septum was found to be deflected to the left after the removal of some of the polyps from the left naris, and a resection of the septum was necessary before the left ethmoid cells could be exenterated. The tissues were very vascular and did not readily accept cocainization. Convalescence was marked by a rapid reformation of a vascular growth in the posterior ethmoid region, and one month after the first operation this new formed tissue was removed thoroughly and the pathologic report was that it was of an inflammatory nature. One year later patient returned with glowing reports of a healthy winter passed in

Texas and full of appreciation for having spent a year free from sickness. Examination showed reformation in the same region, left posterior ethmoid, and so a third operation was recommended. The reformed tissue was removed and a few remaining posterior cells were exenterated from above and to the side of the sphenoid. The sphenoid opening was enlarged and the partition between the sphenoid and ethmoid cells completely removed. The first two operations required nasal packing for persistent bleeding, but no packing was necessary for the last operation, and her convalescence was uneventful. The tissue which reformed before the second operation had the appearance of malignancy, but the laboratory report of chronic inflammation was sustained by the progress of the case.

Case 19.—Mrs. S., age 72. Chief complaints were a boring pain through the upper jaw, a purulent nasal discharge, and difficulty in mastication. Examination showed the right superior maxillary region to be swollen and the hard palate to bulge on the right side. The right middle turbinate was displaced by a granular mass, which bled profusely when a section was punched out for examination. The laboratory reported the condition as sarcoma, and no operative interference was instituted and the old lady died shortly afterwards. This was a case of malignancy of the ethmoid cells and the maxillary sinus.

Case 20.—Mrs. L. M. D., age 36. Chief complaints were cellulitis of the left orbital tissue and a foul purulent nasal discharge. Past history of severe headaches and pain about the left eye for the past year. Examination showed a fistula below the inner canthus of the left eye with swelling and redness of the orbital tissues The left nares was filled with a foul caseous material. Under ether an external opening was made into the ethmoids by enlarging the present opening, the cells cleaned out as well as possible and a through and through drainage established. Strenuous antisyphilitic treatment was instituted and the parts irrigated with Dakin's solution. The acute symptoms subsided and the external wound healed with a very small scar and no deformity, but the internal destruction continued until there was a perforation of the hard palate and the vomer came away en masse. This case was a gumma occupying the ethmoid cells.

Case 21.—E. A. M., age 3. Chief complaints were nasal discharge and large green foul scabs. The child was anemic and quiet and did not desire to enter into play with other children. Examination showed the middle turbinate to be coated with scabs, and a postnasal discharge filled the nasopharynx. The condition was treated by the usual routine suction and nasal douching for one year with no marked improvement. Then came the interval of two years of war. During this period the home treatment had been continued, but acute colds were numerous and scabs would form upon the cessation of douching. The radiogram showed the ethmoids to be of average size for a child of six years, but cloudy and there was an increased density over the antra. Operation: The anterior halves of the middle turbinates were amputated and the ethmoid cells curetted. At the same time the tonsils and adenoids were removed. Convalescence was slow, but the nasal discharge grew less and the scabs became less frequent. During the following winter there were less acute nasal infections and the child improved.

Case 22.—E. J., Jr., age 8. Chief complaints were recurrent acute infection of upper respiratory tract and a low grade fever for which no cause could be found. The tonsils and adenoids were removed in 1918 by an associate, and the record shows a satisfactory operation, but due to persistent obstruction to free nasal breathing a second operation was advised and performed by another surgeon the following year. No lasting improvement followed the second operation. Examination: The nasal membranes were dirty and coated with a thick mucopurulent discharge, the middle turbinates were of average size and there was a ridge and spur of the septum to the right. The nasopharynx was filled with the same discharge and there was a marked hypertrophy of the pharyngeal lymphoid tissue, especially opposite the soft palate. Suction treatment and nasal douching were advised and some improvement was noted. Operation was considered, but patient passed out of our care and remains a case of chronic ethmoid suppuration, which is causing his temperature and an obstruction to free nasal ventilation. Evidently the ethmoids were the cause of the symptoms for which the unnecessary second operation was performed.

Case 23.—Mrs. W. A. W., age 36. Chief complaints were persistent headaches and postnasal discharge. Past history of several nasal operations, including a submucous resection of the nasal septum and amputation of the right middle turbinate. Examination showed the right ethmoid to be hypertrophied and displacing the stump of the middle turbinate towards the center. The left ethmoids were obscured by the middle turbinate. Operation: Right ethmoids exenterated and sphenoid sinus drained. The left middle turbinate was amputated and the ethmoid cells exenterated, and the sphenoid sinus drained. Convalescence was delayed by a reformation of the sphenoid walls, the right sinus being completely filled. This was removed twice surgically and once with cautery, with no avail. A history of a tumor formation in the scar of her abdominal operation which required radiation suggested the use of radium. A 25 mg. needle was inserted into the sphenoid sinus. Two hours after the initial exposure of thirty minutes her headache was better, so the following radium treatment was administered to the right sphenoid sinus:

Oct. 30, 1920. 25 mg. for 30 mins., brass and rubber screening.

Nov. 8, 1920. 25 mg. for 30 mins., brass and rubber screening.

The following day the opening was large enough to irrigate the sinus.

Nov. 24, 1920. 50 mg. for 30 minutes with same screening.
Dec. 20, 1920. 50 mg. for 30 minutes with same screening.
Dec. 31, 1920. 50 mg. for 30 minutes with same screening.
Jan. 10, 1921. 50 mg. for 30 minutes with same screening.
Jan. 27, 1921. 50 mg. for 30 minutes with same screening.
Feb. 9, 1921. 50 mg for 30 minutes with same screening.
Feb. 18, 1921. 50 mg. for 30 minutes with same screening.

Reaction in tissue and right sphenoid sinus deepening. March 21, 1921, two 12½ mg. needles inserted in the right sphenoid sinus for 40 minutes. April 4, 1921, the above needles for one hour into the same sinus. The left sphenoid cavity is larger than its fellow and the partition is deflected to the right. The sinuses were radiographed with a zinc stearate solution acting as a filler, and the left is considerably larger. The radium would cure up the headache, and finally the right sphenoid became normal in size and shape and is now draining. Radiogram to illustrate measures necessary to ascertain if both sinuses were open.

Case 24.—Dr. A. M. L., age 44. Chief complaints were a chronic nasal discharge and headache. Past history of chronic maxillary and frontal sinusitis, for which a Caldwell-Luc antrum operation and several ethmoid exenterations had been performed. Examination showed a well drained maxillary sinus but a blocked frontal sinus on the same side (left). The remaining ethmoid cells were sclerotic and at operation the opening of the frontal duct could not be accomplished to a satisfactory degree. A second attempt several weeks later by removal of the cells in the uncinate process did not improve the drainage. Radium was tried by using the needles (25 mg. for two hours), encased in wax and retained in close approximation to the anterior ethmoid cells. Two weeks later patient returned with a glowing account of his freedom from headache and the lessening of the purulent discharge. A second exposure of 50 mg. for one hour with the same screening was administered, and upon the next visit to the office, three weeks later, the membrane was cleaner and more vascular and his condition greatly improved.

Case 25.—S. J., age 19. Chief complaints were persistent nasal obstruction and postnasal discharge after several nasal operations, including a submucous resection of septum and a thorough ethmoid exenteration. Three subsequent attempts at removal of the reforming excessive granulations and polyps were of no permanent avail. Seen in consultation with my associate, and advised radium treatment. Examination at this time showed the nasal membrane to be edematous and the ethmoid region refilled with polypoid tissue. As much as possible of this new formed tissue was removed, and three exposures of 50 mg. radium for two hour intervals were tried with only slight improvement. The question of protein sensitization was considered but not isolated. At one of the operations the orbit became filled with a hematoma, which displaced the eye outwards and forwards with no bad result. This was caused by a piece of the lamina papyracea coming away in the biting forceps.

Case 26.—W. P. J., age 60. Chief complaints were a progressive obstruction to nasal breathing until complete obstruction had developed. Examination: Nares filled with polypoid tissue coated with purulent discharge. Operation: Polyps removed and degenerated ethmoid tissue cleaned out. Both sphenoid sinuses drained. Convalescence was marked by profuse discharge and the formation of a thick false membrane, which resisted removal by irrigation, and required separation before it could be removed. These radium exposures of 25 mg. in each naris for one hour and a half with needles cleaned up the tissue and reestablished ventilation. Before each radium treatment the false membrane was removed and the excessive granulations cleaned away with biting forceps.

Case 27.—J. L., age 21. Chief complaints were a lack of concentration, persistent ethmoid infection, causing a cough and elevation of temperature. Examination: Nasal membrane thin and red. Left middle turbinate cystic. Septum, spur to left. Suction was productive of a purulent discharge from ethmoid region. Operation: Submucous resection of nasal septum, amputation of cystic left middle turbinate and exenteration of cystic left ethmoid cells filled with pus. Uneventful until sixth day. Postnasal hemorrhage requiring packing of left sphenoid sinus. Later examination showed cessation of purulent discharge with marked improvement of patient's health and a return of a quicker apprehension.

Case 28.—T. L. W., age 22. Chief complaints were a lack of concentration, due to constant frontal headache and ocular pain. Both middle turbinates displaced centrally by hypertrophied ethmoid cells. Previous submucous resection incomplete, leaving a thickened superior half of septum. X-ray of frontal sinuses small and cloudy, ethmoids clear (in fact, too clear), sphenoid of average size. Operation: Submucous resection completed and exenteration of ethmoids and a double sphenoidotomy. Subsequently sphenoids reformed their anterior walls, which required a second and more complete resection. Convalescence was protracted but satisfactory, and as the drainage and ventilation became established headaches were less annoying and concentration more astute, which allowed the patient to continue the study of medicine.

Case 29.—Mrs. O. M. D., age 54. Chief complaints were occipital headaches, postnasal discharge, obstructed nasal ventilation and progressive deafness. Past history of a chronic nasal discharge and a dermatitis of the external auditory canals. Examination showed the ethmoid cells to be hypertrophied, displacing the middle turbinates towards the median line. Operation: A bilateral exenteration of the ethmoid cells and sphenoidotomy. The septum was so thickened above as to obstruct the removal of the high cells, so a submucous resection of it was necessary before the ethmoids could be exenterated. There was a profuse mucopurulent nasal discharge for the first week, which subsided and, much to our surprise, the dermatitis of the external auditory canals improved and there was also better ventilation of the eustachian tubes.

Case 30.—O. P., age 22. Chief complaints were a persistent discharge from right ear, for which a Heath semiradical mastoidectomy was performed fifteen years ago. An atrophic rhinitis with scabs complicated the aural condition, and was thought to be a prominent factor in the continuance of the discharge. A marked septal deformity to the left interfered with any surgical procedure, so a submucous resection of the nasal septum was first done. The ethmoids were found to be degenerated and were exenterated. The anterior wall of the sphenoid was removed. Convalescence was uneventful until the third day, when a hematoma of the septum was apparent. This became infected and required drainage by a tube. The nasal pathology improved and the purulent discharge and scabs abated and with their departure the aural discharge ceased. The antral cavity is easily inspected and well drained. The membranum tympanum has a large perforation in its inferior third, with limiting adhesions which divide the middle ear into compartments, the lower being a direct drain of the eustachian tube. This case shows the possibility of an aural discharge being caused by suppurative ethmoiditis.

Case 31.—Mrs. T. A., age 35. Chief complaints were rapid loss of vision and an acute pansinusitis. In one week vision was reduced to O. D. 10/200, O. S. blind. There was a marked papilledema with swelling of 3 D. of the nerve of the head and a few small hemorrhages about the disc. Fields as shown (Dr. E. C. Ellett). Examination showed an acute suppurative left pansinusitis. A radiogram revealed the left frontal, ethmoid and antrum to be filled with granulations or pus and the right sinuses to be cloudy but not filled. The sphenoid sinuses were cloudy. Operation: Sphenoidotomy and drainage of both ethmoids. The left antrum was thoroughly drained by a Canfield operation. The pus from the sinuses showed a mixed infection of streptococci and staphylococci. In ten days she counted fingers with the former blind eye. On the tenth postoperative day her vision was 20/30 in both eyes, and before the end of the month 20/20 J. 1 in both eyes. Case was formerly reported by Dr. E. C. Ellett.

Case 32.—Mr. E. P., age 52. Seen in consultation on the fourth postoperative day. Past history: Recurrent infection of the ethmoid sinuses and a history of an old luetic infection. Present history: Four days ago a bilateral exenteration of the ethmoids, after the technic of Mosher, which had progressed normally until this morning, when a headache began about nine o'clock. Morphin, gr. 1/4, was administered for the pain, and patient went into a stupor. Examination, 4 p. m., of the same day (fourth postoperative) did not show anything different from the ordinary postoperative nose of the same period. The general appearance of the patient was that of pneumonia, as there was a cyanosis with Cheyne-Stokes breathing. Sensorium was very cloudy. There was no rigidity of the neck and no Babinski reflex or Kernig's sign. Spinal fluid was withdrawn and was cloudy and under pressure. This showed on smear and culture a pneumococcus. The blood also showed a pneumococcus after twenty-four hour growth. The patient did not arouse and died on the night of the fifth day. Before death the lungs showed the first stages of a pneumonia. This was an example of where the infection extended up into the cranial cavity by way of the lymphatics or blood stream.

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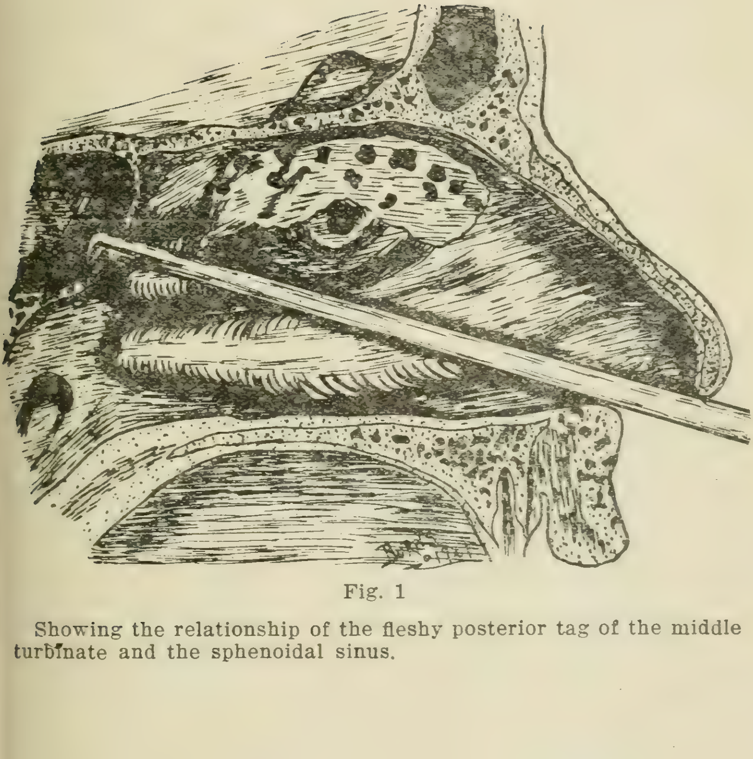

Fig. 1

Showing the relationship of the fleshy posterior tag of the middle turbinate and the sphenoidal sinus.

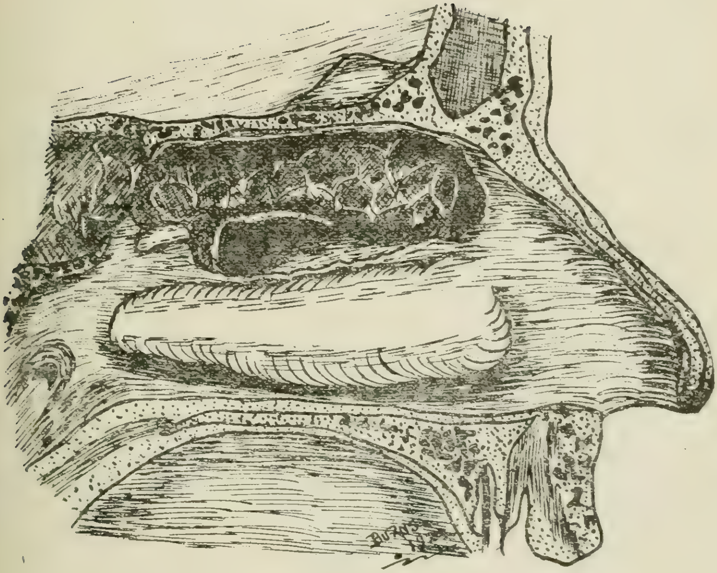

Fig. 2

The complete extenteration and an enlargement of the ostium into the antrum.

XLVII.

THE PRESENT STATUS OF SKIAGRAPHIC INTERPRETATION AS AN ADJUNCT IN THE DIAGNOSIS OF CATARRHAL AFFECTIONS OF THE ACCESSORY SINUSES.*

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I think most of us have long since, as a routine procedure, sent our obscure sinus cases to the roentgenologist for his advice and have largely been guided by his report in our subsequent treatment of that particular case. If, for example, exudate was said to be present in the anterior ethmoid cells of a given side these cells were immediately opened, even though direct examination had revealed nothing abnormal nor had the symptoms pointed particularly to those individual structures. How often at the time of operation have we been disappointed to note the absence of all pathologic changes, and it required a considerable stretch of the imagination to convince ourselves that the operation had really been indicated, much less that the benefits which might accrue would bring about a complete or even partial relief of the symptoms. That we have been placing too great confidence in the X-ray reports, to the detriment of our own better judgment, has slowly been dawning on some of our minds for several years. Levy[1] in an article on diagnosis of sinus disease states that the X-ray is overestimated. LaRue in a discussion of this paper says operation often shows positive disease while the X-rays even negative or even vice versa. Ballenger,[2] in an exhaustive study, says a secondary place should be given to the X-ray and the main reliance should be placed on clinical means.

Boot,[3] while discussing this subject, said many of his reports had been absolutely wrong. Mithoeffer[4] says he considers the X-ray picture as an adjunct of secondary importance.

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*Read before the American Laryngological, Rhinological and Otological Society, 1922.

Culp[5] cites a case in which a prominent roentgenologist, after taking numerous negatives, stated no trouble existed in the sinuses. Shortly afterward she was operated upon and both antra were found filled with cheesy matter and polypoid tissue degeneration. Chase[6] states it is possible to have pus present with practically no shadow on the plate. Guttman,[7] on the other hand, had a case which showed marked cloudiness of the antrum, which on being observed was found to be healthy. Dean[8] writes that radiography is very unreliable in infants and young children. Not so clear as in adults. The sphenoid and posterior ethmoids being almost always blurred on the plates. Long says the X-ray is of considerable service, etc. All these opinions have been expressed within the past twenty-four months. It is my purpose to take these statements as a text for this paper and endeavor to present a frank and unprejudiced discussion of the actual value of X-ray plates to us as rhinologists as an aid to the diagnosis of inflammatory conditions within the sinus cavities.

Let us first briefly consider the standard methods of exposure which are instantly recognized, omitting those which present curious and bizarre shadows making the anatomic orientation difficult for the average laryngologist. The frontal sinus (Caldwell):

1. Posteroanterior. For size and shape; shadow.
2. Lateral. For depth and thickness of anterior wall.

The maxillary. Posteroanterior for shadow and size.

2. Lateral. For relation to teeth.
3. Extended chin (Waters and Waldron). Shadow for comparison with opposite side.

The ethmoid:

1. Posteroanterior. For shadow and extent.
2. Oblique (Rhese).
3. Stenoscopic. For anatomic configuration.

The sphenoid:

1. Lateral. For shadow as compared with ethmoid. Size and shape.
2. Posteroanterior. Shadow.
3. Extended chin. Shadow and size.
4. From above, film in mouth (Pfahler). Shadow, shape and size.

These I think include the methods that today are in general use, the plates from which we are accustomed to examine and confirm the report of the roentgenologist. If we were asked which sinuses in their order were shadows more easily distinguished we would probably say: (1) Maxillary, (2) frontal, (3) sphenoid, and (4) ethmoid. This order would perhaps also check up with our clinical findings as far as confirmation was concerned. Why then do we have such divergent expressions of opinion as to the actual value of radiographs for diagnostic purposes, and why have so many of us been disappointed after confirming our treatment to the X-ray disclosures? Sauer says that improper technic and faulty interpretations are usually the basis of its lack of efficiency, and positive findings frequently depend upon the overzealousness of the interpreter rather than upon the pathologic process. It is said the X-ray never lies. It shows what is there if one can judge by the superadjacent tissues. This, to my mind, is the crux of the entire situation. The shadows must be properly interpreted in order to be of value and, as the personal equation must enter largely into the readings, it is essential that the reader must have checked his experiences with the subsequent surgery, so that his statements as to findings will be reliable data for the rhinologist to unhesitatingly follow. This will undoubtedly occur when the rhinologist himself becomes an expert interpreter of roentgenograms. Before considering the individual sinuses let us for a moment diverge and discuss some of the common causes of errors in interpretation.

1. Faulty Position.—All of us at one time or another have seen ordinary photographs which could hardly be recognized by even a member of the family. Why cannot the same be true of an X-ray photograph? As a matter of fact, it may distort to such an extent that one receives a wrong impression of the plate with a consequently false interpretation. This can be laid to an error in technic and not the reading, but would hardly occur in a well grouped series of exposures.

The presence of air in the sinuses appears to exert considerable influence upon the shadow as to sharpness of outline. Secretion in the sinuses, even thick inspissated, would appear to play a rather unimportant role, although occasionally one could observe a line of demarcation at the fluid level. The shadow, as a rule, however, is not greatly influenced. Swollen and inflamed mucosa gives a denser shadow than exudate and has often been confused with it.

Its relative value in the individual sinuses.

Maxillary.—It is here that the X-ray as a diagnostic measure has its greatest value in sinuology. By means of the anteroposterior exposure comparison with the opposite side is easily and readily made and constitutes the basis of the interpretation, which does not apply so well to any of the other sinuses, not excepting the frontals. Even here, however, the possibilities of an error cannot be overlooked, which may be caused by a shadow due to asymmetry of the bones of the face or the thickening of the lining mucosa in one of the bones of the face or the thickening of the lining mucosa in one of the antra, the result of a former infection.

Frontal.—Much information can be obtained from good skiagraphic plates, provided the proper lines of exposure are observed. The posteroanterior for all around purposes is probably the best, as it not only gives the shape, extent and position of the sinus, but is very liable to give a fairly accurate reading of the condition of the sinus cavity. While shadows were occasionally observed in which no clinical evidence of inflammation was present, on the whole the plates furnished data which could be relied upon and, coupled with the clinical symptoms, were of considerable value in the subsequent treatment of the affection. The lateral position gives information as far as the anatomic configuration of the sinus is concerned, such as its depth, the thickness and position of the anterior and posterior walls, but throws little light on the contents or condition of the mucosa. Skiagraphs of the frontal sinus can then be considered of such value as to warrant them in all cases before operative procedures are applied.

Sphenoid.—Opinions of both the sinuologist and roentgenologist seem to be converging, in that, while these sinuses are distinctly within the scope of the X-ray, it applies more particularly to the anatomic configuration and relations to surrounding parts and less to the actual pathologic condition which obtains within the sinus cavities. The continual experimentation with new positions would seem to lend weight to this assertion.

Cushway,[9] for example, has tried all the various positions recommended and has been extensively experimenting in this direction, but thus far the results had been disappointing. Long,[10] in a comparatively recent writing, considers it of considerable service in demonstrating the size and shape of the sphenoid sinuses, but of limited assistance in giving information as to the gross pathology. As far as the positions of exposure are concerned, there is little to choose between them, as long as there is no anatomic distortion which makes orientation difficult. The anteroposterior position, bringing the sphenoid out through the orbital cavities, seems to have the greatest vogue, generally speaking, but the oblique positions of Rhese and that of Pfeiffer enjoy no little popularity, particularly in the larger cities of the East. None of these methods has proven satisfactory in my own work. I have never been certain that the operation would disclose the precise findings one had been led to expect and have usually not been disappointed. Many times exudate has been positively diagnosed, of which on operation no signs were visible, and vice versa. I have long since discarded the roentgenogram as anything more than a possible confirmatory means to a diagnostic end. Bowen,[11] a roentgenologist, has come to the conclusion that neither the Pfeiffer position nor the reverse one is of much value in the diagnosis of sphenoid sinus empyema. Let us hope the method recently devised by Pfahler,[12] with the film held tightly against the soft palate and the rays directed downward, may prove more reliable.

Ethmoid Labyrinth.—It is here in affections of these cells where most help is needed from the skiagraph that we get the least. When I first had the opportunity to study skiagraphic stenoptican views of the ethmoid, I felt that the X-ray had disclosed its hidden pathology and that hereafter it would be an open book. Subsequent experience has only added to my disappointment, which seems to be shared by many of my colleagues. Thus, Wherry[13] says the field of usefulness of the ray, as applied to the ethmoid heretofore, has been most unsatisfactory. The reason for this is that a shadow or haziness in the ethmoid region does not always indicate an exudate or any change in the cells, as an hypertrophied middle turbinate will give this appearance on the film. Purulent secretion in one or more of the cells will usually not influence the density of the shadow, and even a thickening of the lining mucosa will scarcely affect it. The most reliable indication, from a roentgenographic standpoint, for a pathologic process is the blurring of the intercellular septa. If these cellular walls do not come out clear and sharp in several exposures, undoubtedly a breaking down or a reabsorption has occurred, which may be due to either a hyperplastic or suppurative process. If the absorptive process has caused the formation of one large cavity, the reading will of course be plain, but we are not considering the simple cases, but rather those that are more commonly met with. Summing up, then, the comparative efficiency in the various sinuses, let us arbitrarily place the ratio of value about as follows: Maxillary sinus, 85 per cent efficient; frontal sinus, 75 per cent efficient; sphenoid sinus, 40 per cent efficient; ethmoid labyrinth, 25 per cent efficient.

This does not mean that in a given number of cases this percentage will be found to be absolutely correct, but rather the relative value of assistance the ray gives in disclosing the pathologic condition present in the individual sinuses.

In conclusion, one must frankly state that the mere presence of a shadow in a sinus cannot be considered a reliable indication for operative intervention unless accompanied by the usual clinical symptoms pointing to disease in that particular sinus.

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XLVIII.

OTITIC PYEMIA WITHOUT SINUS THROMBOSIS.

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In 1885 Schwartze[1] called attention to otitic pyemia without sinus thrombosis, which he thought was caused by an osteophlebitis of the mastoid bone. Nothing further appeared in the literature on this condition until Körner[2] published the results of his studies seventeen years later. Although there may be some question relative to the possibility of the production of an otitic pyemia by an osteophlebitis, there can be no question of the value of Körner's deductions of this subject.

He pointed out that this type of pyemia occurred chiefly in children and young adults. It is usually a complication of an acute otitis media or mastoiditis. The metastases were usually found in the joints, serous cavities and muscles; rarely in the lung.

He explained the reason for the infrequency of lung emboli, in that the bacteria are not included in thrombus particles, therefore passing through the lung and, as Wyssokowitsch[23] has shown, lodge in the capillaries of organs in which the circulation is slow—i. e., the spleen, liver, kidneys, bone marrow, etc.

Körner compiled the following statistics, based on 51 cases, many of which were selected from Hesler's[22] compilation on otitic pyemia.

The ages represented were as follows:

14 cases ranging from 0-10 years.
16 cases ranging from 11-20 years.
14 cases ranging from 21-30 years.
5 cases over 30 years.
2 cases, the age not given.

The outcome of these cases was tabulated as follows:

14 were cured by a paracentesis or no treatment.
17 were cured by a simple mastoid operation.

2 by a simple mastoid, plus laying bare the sinus.

5 cases required a ligation of the jugular.

The mortality was 25.5 per cent, or 13 cases. The causes of death were given as follows:

In eleven cases there was a phlebitis of sinus or vein.

In one case there was no infection of the vein or sinus.

In one case the cause was not given.

The prognosis is therefore more favorable than in true sinus thrombosis.

In addition to the production of this type of pyemia by an osteophlebitis, it has been shown that the same condition may be brought about by bacteria passing directly through the sinus walls, or even through the veins and lymphatics of the middle ear.

The above statements have been amply borne out by the case reports of Uffenorde,[3] Breiger,[4] Urbantschitsch,[5] Brühl,[6] Wyssokowitsch,[7] Politzer,[8] Mueller,[9] Rimini,[10] Heine,[11] Schwabach,[12] Zufal,[13] Gruenert,[14] Kobark,[15] Heymann,[16] Massier,[17] Luc,[18] Huyer,[19] Zaalberg,[20] Kopetsky,[21] Hessler,[22] Wyssokowitsch,[23] Kopetsky,[24] Shambaugh[25].

Bacon,[26] Page,[27] McKinney,[28] Boot,[29] Phillips[30] and Oppenheimer[31] have reported cases which agree with the clinical picture described by Körner, but no mention of this is made by the authors.

The two cases reported by Dr. Page are rather characteristic:

Case 1.—A child, two years of age, had a double otitis media following influenza. During her illness her temperature would rise as high as 104 to 106 degrees F., daily. Dr. Page performed a double mastoid operation, but found little destruction of the mastoid cells. Both sinus walls had a dirty grayish appearance, especially the left, but no thickening or other signs of thrombosis were present. He did not open the sinuses. The temperature came down gradually. Eight days later the gauze drainage was removed on the left side; this allowed the soft parts to lie against the vein for twenty-four hours. During this time her temperature came up to 105+. After separating the soft parts from the vein wall and wiping the wound with 5% dichloramin T, the temperature became normal.

Case 2.—A boy of eight years of age was sent home from the hospital on the tenth day following a mastoid operation. In this case the sinus had also been exposed. Wound repair was slow and the sinus had not yet been covered with granulations. He suddenly developed a temperature of 105 F., and was sent back to the hospital. His wound was cleaned up and rubbed with a dichloramin T solution, following which his temperature returned to normal the next day, his white count remaining high for several days.

Some of the conclusions drawn by Heymann[16] are of importance in the development of a paper on this subject. His results after infecting the superior longitudinal sinus of twenty dogs and the lateral sinus of six monkeys by placing a tampon soaked in a culture of streptococci or staphylococci in contact with the sinus wall for varying lengths of time show that:

1. The mere application of bacteria or rubbing them into the sinus wall or even when the wall was scarified, plus the application of germs, did not cause the formation of a thrombus.
2. Pressure of a tampon on the sinus wall also produced no results.
3. When the tampon was saturated with bacteria, thrombosis did occur.

Therefore in a perisinus abscess containing virulent bacteria we have conditions favorable for the production of a thrombus.

4. It is not necessary to have a thrombus to produce an otitic pyemia. The germs may pass directly through the sinus wall. Heymann was able to demonstrate the durchwanderung of bacteria through the sinus wall to the inner side of the endothelium. This durchwanderung may also occur through the walls of the small veins of the middle ear. In several instances he found parietal thrombi which were not infected, but the adjoining wall showed germs penetrating through its inner layers.

Weigert also noticed the einwandurung of bacteria in the veins of the mastoid.[32]

The following case agrees, on the whole, with Körner's osteophlebitic pyemia:

Mrs. S., age twenty-seven, was referred to me by her family physician. She had been suffering with severe pain in the right ear for about one week. Her drum had been incised and the ear was draining freely. She developed a severe pain in her mastoid process, with some edema and tenderness, two days before coming to me, and her doctor was obliged to use opiates to relieve her pain. The patient was undernourished. Examinations of her heart and lungs were negative. Her temperature on admission to the hospital was 101.2 F. Urine examination was negative. White blood count was 10,500. A smear from her ear showed a streptococcus. Examination of her ear showed an edema over the mastoid process, with considerable tenderness to pressure over the tip and antrum. There was considerable dropping of the posterior superior canal wall.

A simple mastoid operation was performed at once. The operation revealed considerable destruction of the mastoid cells. The sinus was not uncovered. Six days after her operation she developed a severe follicular tonsillitis and immediately following this an otitis media of her left ear. The drum was incised, but notwithstanding the fact that the drainage was ample and that every precaution was taken a mastoiditis developed on the left side. A smear showed that we were again dealing with a streptococcus infection.

Several X-ray pictures showed an increasing involvement of the mastoid cells. Again the patient suffered with violent pain, high temperature, edema and tenderness over the mastoid. The left mastoid was therefore opened two weeks after her first operation. Pathologically we were dealing with a hemorrhagic mastoiditis, streptococcus type.

There was not much destruction of bone, and the sinus was not laid bare. Her convalescence now progressed favorably until five weeks after her first and three weeks after her second operation, when on May 25th, I was called to her home, to which she had been discharged from the hospital. I found her in bed bathed in sweat. Her temperature was 106 F., this following a chill, which I was told lasted for twenty minutes.

She was readmitted to the hospital that evening. An internist was called. Erysipelas and tonsillitis were excluded. The lungs and heart were negative. Laboratory tests failed to show the presence of malaria plasmodia. The Widal test was negative, as was the Wassermann. Her white blood count was 10,400. Urine examination showed only a trace of albumin. A blood culture was taken, which later also proved to be negative. The patient's general condition was below par. There was delayed healing of both mastoid wounds; both ears were dry. She had a severe frontal headache and complained of pain over her mastoids and neck, especially on her right side.

On the morning of May 26th, her temperature was 102.2 F., pulse 132, respiration 24. She had another chill twelve hours after being admitted to the hospital. The third chill followed twelve hours later.

About this time she developed an arthritis in her left ankle, which became quite red and swollen. The patient continued to have chills and sweats on May 27th, 28th, 29th and 30th. On May 29th another blood culture was made, which also was negative. A white blood count showed 9,800. This day she developed an arthritis in her left hip, the swelling and pain in the ankle having improved in the meantime.

On June 1st we decided to do an exploratory operation. There were no localizing signs or symptoms, excepting that the patient suffered more pain on the right side. This side was therefore opened first. Here we found an extension of the bone involvement posteriorly over a considerable area with partial destruction of the bone. The sinus was uncovered over a large extent. Its appearance was normal in every respect. The left mastoid was now reopened, but no pathologic conditions were found in the mastoid or in the appearance of the sinus. In view of the fact that it would be extremely hazardous to open both sinuses, we determined to puncture them and, if negative, culture the blood so obtained.

This was carried out after thoroughly sterilizing the mastoid wounds. Both sinuses bled very freely upon inserting the trocar. The bacteriologic examinations of both cultures was negative. The postoperative history is as follows:

The patient had no more metastasis after her operation. Her chills continued every twelve hours up to June 9th, excepting on June 6th, on which day she had but one chill in twenty-four hours. The arthritis cleared up in about ten days following the operation. Her regular temperature continued until June 13th.

CASE SUMMARY.

In a patient convalescing from a double simple mastoid operation, there suddenly developed chills, fever and sweats. Typhoid, malaria, erysipelas, tonsillitis, syphilis and the heart and lungs were ruled out as causative factors. The leucocyte count was never over 10,400, with the blood culture repeatedly negative. Under observations the patient developed an arthritis of the knee and hip of the left leg. She had continued severe headaches, especially on th right side, with tenderness on the same side. Fundus findings were negative.

Reopening mastoids disclosed an extension of the involvement of the right mastoid posteriorly. Both sinuses appeared normal and bled freely upon the insertion of a trocar. Cultures inoculated with this blood proved sterile.

CONCLUSIONS.

I feel that the above findings warrant a diagnosis of otitic pyemia without sinus thrombosis. The causative factor being located in the area of diseased bone posterior to that originally removed from the right mastoid; notwithstanding the fact that the differential diagnosis between pyemia with and pyemia without sinus thrombosis is at present extremely difficult. The main difficulty lies in the fact that it is very hard to rule out the presence of a parietal thrombus.

TREATMENT.

There are a sufficient number of cases of this type reported in the literature to warrant them some consideration relative to their treatment. What form of treatment should we adopt in a case presenting the symptoms of an otitic pyemia, particularly in a younger person, where the sinus shows no abnormality, and especially in the absence of a perisinus abscess? Should we not pause before proceeding with ligation and opening the sinus at once, even though there be a possibility of a thrombus? Would it not be advisable to thoroughly clean up the mastoid and lay bare the sinus, waiting at least forty-eight hours before proceeding with sinus ligation? If the sinus looks sufficiently suspicious one may use a trocar, even though the results may lead one astray where a parietal thrombus exists. If we proceed wth the radical operation unnecessarily we have produced an unsightly scar. We may bring about an extension into the petrosal sinus. Death may supervene due to an insufficiency of the opposite vein. Neither are we certain that emboli may not continue to be thrown into the system through the circulation of the opposite side. If we are dealing with a bilateral affair under the same conditions there is even more reason for treating this condition with conservatism.

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XLIX.

REPORT OF A CASE OF THROMBOSIS OF THE SUPERIOR PETROSAL SINUS OF OTITIC ORIGIN, PRESENTING A SYMPTOM COMPLEX STRONGLY SUGGESTING THE DIAGNOSIS.*

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In considering the diseases of the sinuses of the dura mater secondary to otitis, one finds the literature relating to purulent inflammation of the sigmoid sinus rich in the reports of cases diagnosed and operated with curative results.

Zaufal[1] was probably the first to suggest the operative treatment of sinus thrombosis. With the addition of ligation of the jugular vein and the use of the jugular skin fistula, as practiced by Alexander, the percentage of recoveries is now about 75 per cent.

The symptoms of sigmoid sinus thrombosis are fairly well defined and generally the diagnosis is made without difficulty.

Unfortunately phlebitis of the other sinuses is not so easy of diagnosis. Possibly the lack of clear cut symptoms may account for the sparsity of the literature on primary petrosal sinus thrombosis. Quite a few cases of primary sigmoid sinus thrombosis with extension into the superior and inferior petrosal and even the cavernous sinus have been reported in the literature.

In those cases of extension into the petrosal sinuses the symptoms most frequently observed are swelling of the veins in the temporal region; thrombosis of the retinal veins (Politzer[2]); attacks of epistaxis and epileptiform seizures. When the process has invaded the cavernous sinus a clot will occasionally form in the ophthalmic vein, when there will occur engorgement of the vessels of the orbit, disturbances of vision, photophobia, paresis or paralysis of the motor oculi, abducens and trochlear nerves; exophthalmus, ptosis, edema of the eyelids, neuralgia of the trigeminus, and rarely, sloughing of the orbital tissues[2]. When this symptom complex occurs the diagnosis of thrombosis of the cavernous sinus may readily be made; however, at this stage operative measures promise but little.

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*Read before the Philadelphia Laryngological Society, Feb. 7, 1922.

Thrombosis of the superior petrosal sinus as a primary condition occurs, perhaps, oftener than has heretofore been suspected, but for lack of conclusive diagnostic symptoms it has remained unrecognized. Recently the opportunity was afforded of observing a case of primary superior petrosal sinus thrombosis with extensions posteriorly into the sigmoid sinus, and anteriorly into the cavernous sinus with fatal termination. The symptom complex presented by the case when first seen permitted of but one diagnosis. The case was twice operated, in spite of which the life was not saved. At autopsy the clinical diagnosis was confirmed. It is because of the above facts that the case is reported.

History (as related by patient).—Family history not obtainable in detail, but so far as could be ascertained it is negative as to the present condition.

Past History.—Patient states that he has always been in good health. Two years ago, following an abscess of the left ear, a chronic discharge resulted. At the same time there was an abscess on the back of the neck (apparently a carbuncle). The ear was poulticed and discharged. At first the discharge was odorless, but later became offensive. He has never had bleeding from the ear.

History of Present Illness.—Patient states that up until about three weeks ago he enjoyed good health, with the exception of the intermittent discharge from the left ear. At this time he developed a "cold on the left side of the face," with severe pain extending up over the region of the left temple, along the lower jaw and over the face. (In other words, that region supplied by the trifacial nerve.) The pain was very severe over the side of the head in front of the ear. Patient states that he does not have headache, nor has he had headache at any time during the past three weeks. He claims that at times he has felt chilly, developing into a feeling of warmth and fever, followed by sweat. His answers are prompt and intelligent.

Examination.—The patient appears rather thin, considering the relatively short duration of his illness.

Nose.—Externally the nose is inclined to the right. The nasal septum is deviated high up to the left and low down to the right. There is a bilateral spine along the suture line. The left middle turbinate is visible before shrinking, but is in contact with the septum and with the lateral wall. The inferior edge of the left inferior turbinate is hyperplastic. The right middle turbinate is not visible before shrinking. The inferior edge of the left middle turbinate is hyperplastic.

Throat.—Secondary catarrhal pharyngitis. Tonsils medium sized and submerged; no deposit on pressure.

Functional hearing test revealed moderate reduction of hearing on the left side.

Otoscopic.—A. D. Membrane intact, rather dull, opaque; long process of the anvil not visible because of the opacity. Hammer handle slightly foreshortened. Short process of the hammer quite prominent. Very limited motion with the Siegel otoscope.

A. S.—The greater portion of the membrane has been destroyed. Granulations on the inner wall are visible through thick yellowish purulent secretion, which has an offensive odor characteristic of diseased bone.

Examination of Cranial Nerves.—I, II, III, IV, VI, VII, IX, X, XI, XII nerves on both sides appear and perform normally. The V nerve on the left side presents neuralgia according to the history, but is normally sensitive to all stimuli. The VIII acoustic branch is normal (the hearing impairment was due to the middle ear suppuration on the left side.) The vestibular branch is normal on both sides.

Kernig's sign negative; Babinski is negative; knee jerks perhaps a trifle exaggerated, but bilaterally equal. No ankle clonus.

Further Examination.—Patient has a spot of tenderness to deep percussion about 7 cm. posterior to a line perpendicular to the outer canthus of the left eye and about 7 cm. above a line drawn from the outer canthus of the left eye to the upper attachment of the auricle. The spot itself is about 3½ cm. in diameter.

EYE FINDINGS.

External Examination.—Eyes move well and together in all directions, with nystagmic jerkings when looking to the extreme left and when extreme right. No nystagmus when looking straight ahead.

Pupils round, black, equal in size; react promptly to light, accommodation, convergence and consensually. Vision O. D. 6/6, O. S. 6/6.

Direct Ophthalmoscopic Examination.—O. D. Disc round, sharply defined with the exception of the nasal margin, which appears slightly fuzzy, but within normal limits. Physiologic cup medium size, well defined; lamina cribrosa visible; size and distribution of the vessels normal; fundus, including the macular region, normal.

Direct Ophthalmoscopic Examination.—O. S. Disc round; physiologic cup medium size, sharply defined; size and distribution of vessels normal; fundus, including the macular region, normal; no evidence of choking or inflammation of the disc.

Roentgenographic Findings.—Stereographic roentgenograms made of the head in both right and left lateral positions reveal a shadow on the left side beginning at the frontoparietal suture about midway between the vertex and base, posterior end about one inch wide and the anterior end about one-half inch wide. Irregular in shape and extending forward about 2 inches from the frontoparietal suture and is suggestive of abscess formation.*

Blood Count.—Hemoglobin, 80 per cent; erythrocytes, 4,500,000; leucocytes, 10,000.

Urine.—Amber; acid; sp. gr. 1025; clear; faint trace alb.; sugar, quantity neg.; diazo, acetone neg.; casts neg.

Crystals, leucocytes, erythrocytes, bacteria, epith. cells, normal.

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*Because of the misinterpretation of the roentgenograms, the name of the radiographer is withheld.

Spinal Fluid.—Clear, normal pressure; reaction alkaline; sp. gr. qs. not suf.; globulin neg. to ammonium sulph.; cell count 8 per cm.

In discussing the case with the patient's family physician, Dr. C. F. Hadley, of Camden, Dr. Mackenzie stated positively that the case was not one of brain abscess as was suggested by the X-ray specialist, besides, an exact diagnosis of the case would need to satisfy the following history and findings:

1. An illness of comparatively sudden onset.
2. Agonizing pain over the region supplied by the trigeminal nerve without headache.
3. Fever characteristic of pyemia.
4. Emaciation and pallor (without icterus).
5. Discharge from the ear with the charactertistic odor of dead bone.
6. A blood count showing leucocytosis.

The diagnosis of primary thrombosis of the left superior petrosal sinus of otitic origin was made by Dr. Mackenzie as the only condition that could satisfy the totality of symptoms and findings enumerated above. Operation was considered imperative and was accordingly done.

The patient was operated the same day, November 27, 1921.

The usual postauricular incision through the skin, soft tissue and periosteum was made. The periosteum was elevated from the underlying bone down to and including the tip. The membranous external canal was separated from the superior, posterior and inferior walls of the osseous canal to the tympanic membrane. The mastoid process was opened with Alexander No. 10 chisel and was found to be generally eburnated. The antrum was opened and the bony ridge protecting the facial nerve was levelled to the floor of the canal. The lower osseous wall of the canal was levelled to the floor of the tympanum. The upper wall of the canal levelled to the tegmen tympani. The inner wall of the antrum was necrotic. This necrotic area was evident in the triangle bounded posteriorly by the anterior margin of the sigmoid sinus, superiorly by the tegmen antri and anteriorly by the external and superior semicircular canals. The necrotic area led toward the edge of the petrous bone where the superior and posterior surfaces meet—that is, the location of the superior petrosal sinus posterior to the osseous labyrinth. The inner table of the mastoid overlying the sigmoid sinus was intact and to all appearances normal. The middle ear cavity was carefully curetted and a remnant of the hammer removed. The eustachian tube was curetted. The healthy bone overlying the sigmoid sinus was then removed and its surface appeared normal and glistening; however, following finger compression the sinus tended to refill with blood unusually slow, the sinus with its contents had a putty like resistance. It was then decided that there must be a thrombus in the sigmoid. The deep jugular was then ligated with two ligatures and divided between the ligatures. The sigmoid sinus was next incised, but without a flow of blood. The incision was enlarged and a red thrombus was uncovered. The thrombus was too large and the vis a tergo was insufficient to expel it. For this reason a horizontal incision was made through the scalp and periosteum extending from the posterior margin of the original external incision backward about two and a half inches. The underlying bone was bared and a groove in the bone was cut with the large Luer bone forceps for a corresponding distance. The lateral sinus was exposed half way to the torcular and the original incision of the sinus was extended posteriorly another inch and a half. A small spoon curette was introduced into the sinus posteriorly to the torcular when the thrombus was removed quite intact. Iodoform gauze pack was introduced into the wide open lateral sinus. Plastic after Panse. Iodoform gauze wicks were introduced into the wound cavity; outer plain gauze dressings and bandage were applied. The fact should be emphasized that there was no erosion of the inner cortex covering the sigmoid sinus. Neither was any cholesteatoma found.

The left superior petrosal sinus was not opened because the patient was in a very poor condition, and it was hoped that the expelling of the clot from the left sigmoid sinus had served to empty out the left superior petrosal sinus.

On the third day following operation (November 30, 1921,) the patient's temperature had fallen to 98 F., and remained below 98 F. until the evening of December 2nd, when it reached 99 F. The following morning it had fallen to 97.8 F., but reached 100 F. the evening of December 3rd. On the morning of December 4th the first redressing was made, at which time the patient's temperature was 99 F. At 2 p. m. the same day the temperature was 98.2 F., but by 5 p. m. it had risen to 101 F. and at 10 p. m. it was 101.6 F. No chill was reported by the patient, nor was any noticed by the nurse. Aching of the teeth on the left side was complained of. There was no headache; no changes in the eye grounds observable at the bedside with the electric ophthalmoscope. Babinski was negative. There was no spontaneous nystagmus when looking straight ahead. The spinal fluid was normal. The blood Wassermann was negative, as was also the spinal fluid. The leucocyte count was 11,000.

December 7th, following the third redressing, the patient developed a chill with a rise of temperature to 103 F., followed by a fall to 99.6 F. The patient still complained of pain in the teeth on the left side. No fundus changes were observed. Babinski negative; spinal fluid normal; leucocyte count, 9,500; urine analysis normal; patient delirious at times.

Dr. G. W. Mackenzie was again called in and he felt that a remaining clot in the left superior petrosal sinus was the cause of the trouble. A second operation was performed on December 9th. The thin intervening wedge shaped piece of bone that remained over the region of the left superior petrosal sinus was removed and the sinus incised. No free bleeding was obtained. The smallest spoon curette was introduced into the sinus and a small clot was removed. A wick of iodoform gauze was placed in the cavity of the sinus. This operation was not followed by any improvement of symptoms.

Ptosis of the left eyelid was first noticed on December 10th. Pupils still equal in size and react normally to light. The patient was delirious at times. The Babinski was questionable on the right side. Small spasmodic twitchings were noticeable on the right side of the face, accompanied by a tendency to grind the teeth. The spasms were tonic in character and seemed to involve the muscles supplied by the seventh cranial nerve. The spinal fluid still remained normal. The mastoid wound looked healthy except for a small area necrosis in the region of the superior petrosal sinus just posterior to the osseous labyrinth and anterior to that which Dr. Mackenzie removed at the second operation. Whether it was present at the time of the second operation or developed later is a question; however, I am inclined to believe it was present at the time, for the operator hesitated to open the labyrinth for fear of labyrinthine infection following and adding another complication. No changes in either fundus are evident at this time (December 13, 1921.) The patient was in very poor physical condition. Nourishment was taken only with the greatest difficulty.

The morning of the 14th of December the first pupillary change was noted, when the left pupil was found to be slightly larger than the right, but still reactive to light. There was also present a slight degree of exophthalmos of the left eye. Spinal puncture made at this time gave a questionably slight increase in pressure; otherwise it was normal. At 2 p. m. the left pupil was dilated and fixed. The left fundus showed blurring of the disc outline. There were spasmodic seizures of the right side of the face and the right shoulder and arm, but not of the right leg. The patient died at 8:30 p. m.

Postmortem findings: Patient, male; emaciated appearance. The left side of the brain beneath the dura felt fluctuating. The dura was incised and a purulent exudate sponged out. There was no evidence of brain abscess. The brain is normal except for those surface changes found in the presence of a circumscribed purulent meningitis. The sigmoid sinus was observed to have been opened during life. The left superior petrosal sinus was partly opened with a red thrombus extending from about its opening into the left cavernous sinus. The left cavernous sinus also contained a red thrombus.

There is no involvement of the sinuses on the right side.

COMMENTS.

1. Given a case presenting the three cardinal symptoms offered by this patient, namely, (a) trifacial neuralgia, (b) pyemic temperature, (c) the presence of a chronic purulent otitis media, with a discharge having the odor of dead bone, the diagnosis of primary thrombosis of the superior petrosal sinus of otitic origin is justifiable.

2. From the experience gained in this case it is felt that in primary thrombosis of the superior petrosal sinus it is far better to sacrifice the labyrinth in the effort to secure a freer exposure of the superior petrosal sinus than to attempt to conserve the labyrinth at the cost of a less free exposure of the sinus.

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ELECTROCOAGULATION IN THE TREATMENT OF CARCINOMA OF THE LARYNX.

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Diathermy, or electrocoagulation, has long been recognized as an accepted therapeutic measure. Nagleschmidt of Berlin, in 1907, demonstrated the use of high frequency currents in the heating through of tissues. But the special application of diathermy in the field of otolaryngology is a matter of more recent development.

Norman Patterson, in the Lancet in 1919, advocates electrocoagulation of malignant tumors of the larynx in preference to other surgical procedures. W. J. Harrison, C. A. Pfender recommend diathermy in the management of laryngeal carcinoma. On the whole, however, the literature is surprisingly meager in this field, and a complete technic has perhaps nowhere been developed and accurately described.

It is not the purpose of this paper to go into the physics of diathermy nor to dwell at any great length upon the technic of its application to the larynx. A few definitions are perhaps necessary. Diathermy is a means of raising the temperature of tissue by the resistance of the tissue to the high frequency, or D'Arsonval current.

Saberton differentiates between medical and surgical diathermy by the fact that medical diathermy aims to raise the temperature of the tissues within physiologic limits, and surgical diathermy aims to exceed these limits and to produce tissue destruction by coagulation.

There are several special problems to meet in treating the larynx and not the least of these is the question of anesthesia. Local anesthesia is not advisable. Block anesthesia is possible, but cannot be recommended. Of the general anesthetics ether cannot be used because of the great danger of explosion from a possible spark from the electrode. Nitrous oxide and oxygen are not sufficient to produce complete relaxation, and there is great difficulty in maintaining deep surgical anesthesia. The socalled synergistic anesthesia used extensively by Beck and others in laryngeal surgery is extremely dangerous in these cases, because of the fact that sufficient ether is excreted by the lung to produce an explosion from the accidental sparking at the tip of the electrode. We have been forced to use chloroform at the County Hospital. This anesthetic is surprisingly satisfactory. Its traditional dangers are in a large measure obviated by having a highly skilled anesthetist administer it. There is no danger of explosion, relaxation is complete, it is rapid and the small amount of it necessary to carry on deep surgical anesthesia makes it highly desirable.

Another problem is that of the method of approach. The tumor may be exhibited by doing a laryngofissure. We have condemned this method because of the possibility of dispersion of metastases by cutting into a portion of the tumor. The indirect method of laryngoscopy is not recommended because precision in the application of the electrode is very difficult. There remains suspension laryngoscopy and that is the method par excellence.

The larynx becomes clouded with dense cloudy vapor from the dessication of the tissues during the operation, and it is difficult to see the field of operation. It has been recommended to change the air in the larynx by blowing a stream of air from a pump, but the method found most satisfactory by us is to place a thin metal tube into the larynx, connect this with the suction pump and the steam from the coagulating tissues is thereby removed from the larynx as rapidly as it is formed.

The problem of dosage is still far from solved. It is better in general to err on the side of not doing enough than to do too much, for the operation can be repeated several times, whereas too extensive a coagulation increases the danger of serious hemorrhage when the slough separates. Specifically for a tumor with a diameter, roughly of three centimeters and using an electrode one centimeter in diameter we apply the electrode in two places. With a current of 1200 to 1500 milamperes the electrode is applied in each place for twenty seconds. It is too early to say whether this dosage is sufficient, but we are inclined to believe that it is not. It will only be possible to determine this by the study of the autopsy and thus far none of our cases has proceeded to that stage. It must be remembered that the average laryngeal carcinoma seen at the County Hospital is far advanced. The early lesion is a rarity. The dosage for a small, early lesion is naturally smaller than that for the lesion which involves the entire larynx. The favorable case is one in which metastastes have not formed and in which the lesion is small.

The immediate results of diathermy are as follows: There is a surprisingly small degree of edema, a preliminary trachetomy is imperative nevertheless. There is a marked and almost dramatic lessening of pain. The bleeding from the ulcer stops. The fate of the coagulated mass is either a sloughing en masse or a fractional sloughing over a period of time. When the coagulation has been radical and extensive there seems to be a tendency to mass sloughing with the attendant danger of severe hemorrhage. When the coagulation has involved only a portion of the tumor, in other words, when the operation has been less radical and when the sloughing has been fractional, the danger of severe hemorrhage is correspondingly lessened, thus making small and repeated dosage more desirable than a complete, radical coagulation.

Electrocoagulation supplemented by radiation is recommended by Pfender. Rhodenburg and Prime have shown that a combination of diathermy (producing a low degree of heat) plus radiation have a greater effect upon malignant tumors than either diathermy or radiation alone. These findings have been confirmed by B. C. Corbus and V. J. O'Connor of Chicago, in the study of the bladder.

Briefly stated, the advantages of electrocoagulation in the treatment of malignant tumor of the larynx are as follows: The operation is bloodless; there is an immediate lessening of pain; there is no danger of dissemination of metastases. The deep penetration of the heat has an inhibitory effect upon the growth of malignant cells (that is, the cells beyond the area of coagulation), as has been shown by Rhodenburg and Prime experimenting with neoplastic cells in vitreo.

Corbus and O'Conor have by animal experimentation found that the body defense against carcinoma metastasis lies in the formation of a connective tissue capsule The scar tissue that is formed after a diathermy burn is more dense and spreads farther into the surrounding tissue than is the case after any cutting operation. We have here then an extensive reinforcement of nature's attempt to throttle the embryonal cell.

To sum up:

1. Diathermy of carcinoma of larynx is a relatively untried measure. It offers a promising field for investigation.
2. It not only coagulates the tumor mass, but has an inhibitory effect upon the growth of malignant cells adjacent to or at an appreciable distance from the tumor.
3. Chloroform is the logical choice of anesthetic.
4. The laryngeal tumor should be approached by means of suspension laryngoscopy.
5. The amount of current—the length of exposure necessary—requires considerable experience upon the part of the operator to determine, because no set of definite rules of procedure can be formulated in the light of the present limited knowledge of the subject.
6. Radical dosage cannot be recommended.
7. The edema following diathermy is surprisingly slight, nevertheless preliminary trachetomy is imperative.
8. Pain is diminished markedly following coagulation.
9. Diathermy should be supplemented by radiation.

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LI.

VASOMOTOR DISTURBANCES OF THE UPPER AIR PASSAGES AND SINUS DISEASE.

By J. L. Goodale, M. D.,

Boston.

In the present paper the term vasomotor disturbances of the upper air passages is used to denote the conditions known clinically as vasomotor rhinitis and asthma.

The term sinus disease is here restricted to those alterations of the ethmoid sinuses and of the maxillary antrum which are characterized by edematous hypertrophy arising independently of dental infection. Involvement of the frontal and sphenoidal sinuses may or may not be present. Clinically we find all gradations from polypoid degeneration of the middle turbinate to advanced polyp formation. The maxillary antrum usually contains secretion, in come cases seropurulent in character, with a predominance of mucin. In more advanced stages of the inflammation the periosteum becomes the site of infiltration, particularly in the region of the middle turbinate, giving rise to rarefying osteitis. Bacteriologic examination of the antral fluid is rarely sterile, at times diphtheroid bacilli, and at others the various cocci, such as the pneumococcus, staphylococcus aureus and albus and streptococcus, being present.

It does not appear as yet definitely established whether an individual experiences sensitization to bacteria in the same manner as to proteids entering the system by inhalation or ingestion.

In 1916 the writer offered evidence to show that some anaphylactic individuals give a positive skin test to certain bacterial proteids and that these proteids differ among themselves in their power of causing the skin reaction. Further observation by others, notably by Walker and Rackemann, indicate that anaphylactic individuals may show a very wide variety of bacterial sensitizations.

In a recent paper by Cooke (Journal of Immunology, March, 1922), doubt is expressed whether the group of socalled infectious asthmas can be recognized as a distinct pathologic entity, and the suggestion is made that these may in reality be the result of inhalation of various hitherto unrecognized forms of dust in sensitized individuals.

Without entering into a discussion of this question, the writer wishes to state that in a fairly large number of cases the correlation of clinical with laboratory data seems to lend weight to the belief that sensitization to bacteria exists, and that this sensitization is responsible for the development of vasomotor rhinitis and for asthma in a certain group of cases.

Even if this point be granted, we are not as yet fully clear regarding the nature of such a baterial sensitization. From the standpoint of systematic botany, we might perhaps conceive it to be a sensitization to the proteid of the bacterial body rather than to the products of metabolism of the organism. On this hypothesis the bacterial proteid would be regarded as analogous to the proteid of pollen and of foods. The microorganisms which are recognized as invaders of the upper air passages in acute infections possess the characteristic in common of exerting their influence through the liberation of an endotoxin—that is, a toxic substance existing within the wall of the organism and escaping when the wall becomes sufficiently permeable. This endotoxin is the agent which excites inflammatory symptoms in the invaded individual and gives rise to the phenomena variously of acute rhinitis, pharyngitis, laryngitis and bronchitis. The anaphylactic individual may owe, however, the occurrence of his particular vasomotor symptoms to the specific proteid of the organism and not to the endotoxin. When he is attacked by one of the organisms in question he experiences first the symptoms of a cold in the head or an acute bronchitis from the endotoxin of the organism in question, but these symptoms of inflammation are more or less completely submerged with the liberation of the bacterial proteid.

If this hypothesis should prove correct, it would perhaps afford an explanation of the immediate skin reactions occurring in some cases on the application of bacterial proteids to a scratch, and at the same time indicate that the reactions occurring after the intradermal injection of vaccines may be a toxic manifestation rather than an anaphylactic one. Improvement observed in the asthmatic symptoms from the administration of vaccines would then be explained as the result of the establishment of increased immunity to the organisms rather than as a desensitizing process.

Cases have come under my observation where asthmatic symptoms follow a certain type of acute catarrhal infection, and are absent in a subsequent attack of acute rhinitis or bronchitis. As an example, a woman with chronic ethmoidal and maxillary sinusitis, came with a history of nearly constant asthma of several years' duration. Radical operation upon the sinuses was followed by relief of the asthmatic symptoms. Subsequently the asthma reappeared in association with certain acute infections of the upper respiratory tract, but it was found that not all acute infections were followed by asthma. Bacteriologic examination of two attacks with asthma showed a sputum with streptococcus, while on three occasions the sputum from acute bronchitis without asthma showed nearly pure cultures of staphylococcus albus.

There are probably few subjects in which the experience of laryngologists is able to furnish a greater abundance of data than in the one under consideration, but also few in which the evidence of statistics can be more misleading. We are dealing with a number of variable factors, such as the degree of development of the pathologic alterations, the kind and number of microorganisms present, these combining in many different ways to exert an effect upon an individual already himself under the influence of other general forces of a beneficial or harmful character.

Furthermore, the operative measures undertaken in a given case may have been faulty or inadequate, with the result of rendering conclusions inaccurate regarding the etiologic relationship between the sinus disease and the vasomotor disturbance. These remarks apply with more force to asthma than to vasomotor rhinitis, in which less complexity of the various factors exists. Instead, therefore, of attempting to offer a statistical review of all the cases which have come under my observation, it has seemed to me preferable to present for your consideration some of the conclusions to which I have been led from a study of patients in private practice, upon which I have operated in recent years, recognizing the fact that these are by no means final but are rather of the nature of suggestions to be criticized and probably modified.

Vasomotor disturbances of the regions under consideration may owe their origin variously to the inhalation of foreign proteids, to the ingestion of foods, to the development within the body of infecting microorganisms or to the influence of reflex stimuli. In a given case the causative factor may be one or more of these agents operating singly or in association. Consideration in the present paper is limited to conditions of apparently infectious origin, or to those in which inhalation and ingestion factors, if present, play an intermittent or subordinate role.

It has not been suggested, so far as I am aware, and my observations do not lead me to believe, that anaphylactic disturbances arising from the inhalation or ingestion of foregin proteids, however prolonged, can, in the absence of bacterial infection, give rise to the pathologic alterations of the sinuses above mentioned. We often, it is true, see a hay fever or an occupational inhalation vasomotor rhinitis, as from horse dander or from flour dust, pass gradually into chronic sinusitis, but careful examination of the patient's history will show a series of attacks of acute rhinitis. Since, furthermore, we cannot distinguish, at least from clinical inspection, between the sinusitis unaccompanied by vasomotor symptoms and that occurring in an anaphylactic individual during the periods between the vasomotor manifestations, it seems reasonable to state that the weight of evidence is at present against the production of sinusitis through the agency of inhaled or ingested foreign proteids.

In relation to the question as to whether sinus disease plays an etiologic role in the production of these socalled infectious vasomotor disturbances, we may leave out of consideration to what extent the sinus disease is itself the result of infection. We are concerned simply with the determination whether sinus disease, however established, initiates or influences the vasomotor disturbances, and consequently whether remedial measures directed to relief of the sinus disease will have a corresponding influence on the vasomotor symptoms.

The essential characteristics of vasomotor rhinitis can be determined with relative completeness. We have a pathologic condition corresponding to the familiar wheal produced by the proteid skin test, namely, a serous infiltration and sensory disturbances. Pallor and swelling of the mucous membrane result, together with an outpouring of fluid, and accompanied by sensations of itching with tendency to sneezing.

These anaphylactic phenomena may occur in nasal mucous membranes which are otherwise essentially normal, or they may be superimposed upon tissues already altered by chronic inflammation. In the latter case, a knowledge of the pathologic conditions will enable us to translate the descriptive clinical terms into their histologic equivalents.

Asthma in association with sinus disease may be acute and recurrent or chronic. In the former type we find the individual living for the greater portion of the year in a state of health, but periodically subject to invasion of his mucous membranes by infecting microorganisms. When this invasion develops we find clinically for a few days the symptoms of a cold in the head or of an acute bronchitis, soon, however, succeeded by symptoms of vasomotor disturbance, showing vasomotor rhinitis or asthma. The duration then of the vasomotor affection is like that of an acute, nonanaphylactic disturbance of the tissues in question, and, after recovery, the patient remains free from symptoms until the next invasion.

In the chronic type the individual suffers more or less constantly from asthma, the periods of freedom from symptoms being either brief or absent. Vasomotor rhinitis may coexist with the asthma, although rarely.

In view of the frequent association of sinus disease and of bronchitis, the question suggests itself whether there is an etiologic relationship in the pathologic conditions existing in the regions which predispose them to invasion.

Webb and Gilbert (Journal A. M. A., 1921, p. 714) report a series of cases of chronic bronchitis, in which examination was made of the maxillary and ethmoid sinuses. In a large parcentage of cases sinus disease was found, and operative treatment of the sinuses brought about marked improvement in the condition of bronchial inflammation.

Mullen (Transactions Laryngological, Rhinological and Otological Society, 1919) has shown in animals that lymph absorption from the antrum, whether of bacteria or inert substances, is by way of the submaxillary and internal jugular nodes to the lymph ducts of the great veins, the right heart and the lungs.

In a paper, "Recurrent Influenza in the Nose and Throat," read before this association in 1910, I called attention to a group of cases characterized by acute, recurrent inflammation of the nose and throat, with secondary involvement of the bronchial tubes. The past history is one of annual attacks of inflammation entering the lymphoid tissue of the pharynx and extending promptly to the ethmoid and maxillary sinuses, with involvement of the trachea or bronchial tubes. Purulent discharge appears in the nose and simultaneously cough with purulent expectoration. Examination of the chest shows well defined bronchitis.

If the group of symptoms above described occurs in an anaphylactic individual the symptoms of inflammation may be accompanied by manifestations of asthma which dominate the clinical picture.

It does not appear possible to determine in a given case the relative amount of absorption of bacterial proteids from the mucous membrane of the sinuses and of the bronchi respectively. We may be able to form only an approximate estimate derived from clinical data concerning the degree of development of the sinus disease, the extent of the bronchitis and existence of bronchiectasis. In advanced cases of bronchial disease, especially associated with myocarditis, it would seem of doubtful value to perform a radical operation on the nasal sinuses solely for the purpose of restricting the amount of absorption of bacterial proteids.

The operative management of these conditions of sinus disease in anaphylactic individuals does not differ from that in the ordinary case. It is, however, of especial importance to determine with accuracy the condition of the antra. Evidence obtained from transillumination and X-ray may be misleading, since these methods may show simply the existence of a disparity in the illumination of the two sides. It is possible that one antrum may be opaque and the other relatively clear, but nevertheless both may have fluid in the interior.

One side may be in a more advanced stage of inflammation with greater thickening of the mucous membrane than the other. In all the cases which I see now, when I have reason to suspect the existence of sinus disease, I do a paracentesis on both sides and have repeatedly found fluid in the antrum which had been pronounced normal by the X-ray.

The antral opening should be enlarged to avoid any possibility of closure and to permit the free escape of the frequently thick viscid fluid. A larger opening is required in these conditions than in the case of simple empyema of the antrum, where the fluid is thinner and more readily capable of flowing out through a small opening.

If the ethmoid regions are affected it is desirable to remove the anterior end of the middle turbinate and curette the anterior ethmoid cells.

Results of operative treatment in chronic vasomotor rhinitis of the type under consideration have been satisfactory in the larger proportion of cases, and the patients have remained free from symptoms except in the event of their experiencing a subsequent attack of acute infection, when a transitory form of vasomotor disturbance may appear. Such a condition is illustrated by the following case:

Mrs. R., 35, was seen November 18, 1920, with a history of vasomotor rhinitis, of one year duration, following a severe cold. Examination showed general pallor of the mucous membranes of the nose, with polypoid degeneration of both middle turbinates. Transillumination was dark on right. All skin tests for foods, pollens and dusts were negative. Both antra were opened through the lower meatus, and the ethmoid cells curetted. The right antrum was filled with thick viscid mucus which showed numerous colonies of staphylococcus albus and ozena bacilli. The vasomotor symptoms disappeared after the operation. One year later the patient had an acute rhinitis, accompanied by vasomotor symptoms, the whole process clearing up, however, within a few days.

Four other cases of essentially similar type have been operated upon in the past two years, with the result of definite relief of the vasomotor symptoms.

In eighteen patients showing sinus disease associated with asthma, of the type under consideration, results of operative treatment have been beneficial, so far as the asthma is concerned, in the majority of the cases. Sometimes after the operation the asthma subsides or remains absent for a while, but is apt to reappear later. Some unsatisfactory results have been manifestly due to imperfect operative technic in the sinus operation, where fluid has reaccumulated in the antra. Washing out the fluid has been followed by again a relief of the asthma. A formation, however, of a large antral opening, which requires no washing, has been followed by a larger number of successful results. In a certain proportion of the cases the change in the clinical picture has been from chronic persistent asthma into an acute recurrent asthma, the patient remaining for a much greater time free from disturbance, but on the development of a cold or acute bronchitis, or after other forms of reflex strain, again experiencing attacks. It has seemed to me probable that the improvement in the chronic asthma is due chiefly to a lessening of the bronchitis and consequent diminution in the amount of bacterial proteid absorbed from the bronchial tract. This view is in harmony with the observations above mentioned regarding the relief of bronchitis by the eradication of sinus disturbance.

My experience with vaccines in these cases of vasomotor disturbance with sinus disease leads to the conclusion that they are of relatively little value so long as the diseased state of the sinus persists. It is only after clearing out the pathologic disturbances in the ethmoidal and maxillary sinuses with the establishment of permanent free drainage in the latter that they are of service.

Where, however, this has been done, the administration of autogenous vaccines, which by the intradermal test excite definite reaction, has appeared to exert a material influence upon the asthmatic symptoms.

In conclusion it must be emphasized that the management of asthma with sinus disease is not alone a problem for the laryngologist but requires the close cooperation of the internist, with constant supervision by the latter of the patient.

Operation on asthmatics with sinus disease should not be undertaken until all possible extrinsic causes, as from dust and foods, have been eliminated. It happens not infrequently that a case which appears to belong to this category of infectious asthma may in reality owe the disturbance to the presence or a cat or dog in the house or to some article of food in the diet. The observations of Cooke on the influence of house dust are of great importance in this connection.

The degree of development of the bronchitis, the presence of bronchiectatic cavities and association of myocardial affections of the heart must be taken into consideration. If the pulmonary lesions have advanced, little or no benefit may be obtained. In patients with myocarditis an operation may be contraindicated, or at least may be considered likely to afford little or no benefit. As an example, H. B. developed ten years ago asthma and bronchitis following influenza, with annual attacks of bronchitis subsequently. In October, 1921, the asthma became more pronounced, and there was evidence of beginning myocarditis. Both antra were dark on transillumination, and considerable mucopus was present in the nose. Extrinsic causes of asthma were eliminated. Findings were typical of infectious asthma, and an operation would have been performed, except for the condition of the heart, which showed signs of rapidly increasing weakness. The patient died in January from heart failure.

REPORT OF CASES.

1. M. A., 7.—Asthma, four years' standing, associated with frequent colds and attacks of bronchitis. Negative to foods and pollens. Right maxillary and ethmoidal sinusitis. Chronic tonsillitis. Antral fluid showed staphylococcus albus and streptocuccus pyogenes. Radical operation on sinuses. Asthma disappeared and remained absent for nine months, but reappeared on taking cold, lasting moderately for two weeks. Two attacks of asthma occurred during the following year in association with acute infection. No trouble during last six months. Chest now clear.

2. Mrs. E. F. S., 35.—Chronic asthma and bronchitis many years' duration. Marked maxillary and ethmoid sinusitis. Radical operation on sinuses. Staphylococcus aureus in antral fluid. Asthma and bronchitis gradually cleared up. Has been well during past year.

3. W. A., 50.—Asthma and bronchitis 15 years' duration, following severe cold. Ethmoidal and maxillary sinusitis. Operations on ethmoid done repeatedly without relief to asthma. Autogenous vaccine from staphylococcus albus and streptococcus given without result. One year ago both antra opened, left showing considerable mucopus. Bronchitis has improved and for past year has had no asthma.

4. S. W. M., 69.—Asthma many years. Chronic maxillary and ethmoidal sinusitis. Much viscid mucus removed from right antrum showing staphylococcus aureus. Gradual improvement in bronchitis with diminution in wheezing. No symptoms during past year.

5. Mrs. F. W. N.—Asthma many years, recurrent. Maxillary and ethmoidal sinusitis. Radical operation. Improvement in asthma and bronchitis during following three months. Remained then free from asthma until streptococcus bronchitis one year ago, lasting one month. Since then free from disturbance.

6. M. A., 40.—Asthma several years with bronchitis. Many operations have been done for removal of polypi. Marked inflammation of ethmoid bones, opacity over antra. Radical operation on sinuses. Slow improvement during following six months, attacks of asthma, however, being less frequent and severe. Seen two years after operation, having had no asthma during previous year.

7. Mrs. G. E. K.—Thirteen years ago chronic right maxillary and ethmoid sinusitis observed. Radical operation done. Now right frontal sinus shows considerable secretion. Intranasal operation on right frontal sinus. Four months later asthma began gradually, increasing, in association with involvement of left antrum. Antral fluid showed staphylococcus aureus and albus. Autogenous vaccines from these followed by no relief. During following year asthma became more trodblesome, and infection of right antrum was observed. In May, 1920, both antra were opened freely, and polypoid enlargement of left middle turbinate removed. Has had no asthma for two years since operation.

8. Miss E. F. M., 70.—Seen first fourteen years ago. Asthma many years' duration. Repeated operations on ethmoid sinuses without relief. Sinuses were then curetted with-

out improvement of asthma. During next eight years no essential change in asthma; chronic bronchitis was noted in 1913, the right antrum became infected, and was tapped with removal of much pus. Autogenous vaccines were administered without relief. Five years ago tonsils were removed and showed large amount of detritus. Chest examination showed extensive chronic bronchitis with a moderate degree of myocarditis. Asthma still intermittently troublesome. No definite result could be shown following the various operations.

9. D. D., 30.—Bronchitis two years following severe strain during war. Marked ethmoid sinusitis. No involvement of antra. Curettage ethmoids was done nine months ago. Antra opened. No fluid found. No relief following operation.

10. Mrs. C., 50.—Seen 17 years ago. Asthma many years' standing with chronic bronchitis. Marked ethmoid sinusitis. In 1906 large amount degenerated ethmoidal tissue curetted on both sides. Asthma has persisted more or less constantly since. It has not been possible to obtain the consent for operation on antra, although these are evidently infected, as shown by X-ray. Now has extensive bronchitis with myocarditis.

11. Mrs. C. H. M.—Asthma ten years, more or less constant. Some operation on sinuses was done ten years ago, with removal anterior ends middle turbinates. Transillumination at time of examination dark both sides. Curettage of ethmoid cells with large opening into each antrum. Antral fluid showed streptococcus. Asthma has been troublesome since operation during past six months following operation, but as patient lives at a distance it has not been possible to follow condition accurately.

12. Mrs. E.—Seen August, 1914, history of infection right antrum last spring, with asthma more or less constant, conditions persisting throughout summer and winter with each cold. In August, 1915, both antra were tapped, large amount of fluid removed, asthma nevertheless persisting. In October antra were again opened, and large amount viscid mucus removed from left. Middle turbinotomy. Improvement followed lasting several months, asthma again returning. Sinuses repeatedly washed with temporary relief. In 1916 no asthma throughout summer. Both antra free. In winter of 1916

asthma reappeared, with evidence of myocarditis. In January, 1916, large free openings made into each antrum, which showed a quantity of viscid mucus. Cultures of this fluid showed diphtheroid bacilli, few streptococci and occasional colonies of staphylococcus. Asthmatic attacks persisted throughout winter of 1918 and 1919, becoming extremely severe in March, with progressive heart failure, from which the patient died March 16th.

13. Mrs. G. Y. B., 40.—Asthma and chronic bronchitis many years. Chronic tonsillitis Chronic ethmoidal and maxillary sinusitis. Tonsillectomy and radical operation on sinuses. Staphylococcus albus in antral fluid. Asthma disappeared, remaining absent for three months. In past six years, five short attacks of mild acute bronchitis and asthma.

14. Mrs. A. P., 35.—Asthma several years, generally persistent. Chronic ethmoidal and maxillary sinusitis. Streptococcus in antral fluid. Radical operation. Asthma became progressively less severe during following two years. For past three years no asthma.

15. Mrs. J. B. P., 35—Asthma several years, generally persistent. Chronic bronchitis Chronic ethmoidal and maxillary sinusitis. Antra opened. Cultures of antral fluid showed staphylococcus albus and streptococcus pyogenes. Since operation five years ago attacks of asthma infrequent, usually brief and following colds or overwork.

16. Mrs. M. F. R.—Frequent attacks asthma and bronchitis. Chronic ethmoidal and maxillary sinusitis. Radical operation. Since operation three years ago no asthma.

REMARKS.

In all the above cases skin tests for foods and pollens were negative. A few showed positive reactions to dusts such as horse dander and cat hair, but the influence of these factors upon the asthma was definitely eliminated.

The statement, "no asthma," denotes that the patients have been free from symptoms of the type which had previously troubled them. Excessive fatigue, nervous strain or exposure to certain dusts, in patients sensitized to such proteids, have been sufficient to excite a temporary recurrence of the asthmatic symptoms.

CONCLUSIONS.

A group of vasomotor disturbances characterized by vasomotor rhinitis or by asthma exists standing in relation to chronic infection of the nasal sinuses. In vasomotor rhinitis the sinusitis is probably the direct cause of the disturbance. In asthma of this type, it is not wholly clear what part in the production of the disturbance is played by the presence of bacterial infection in the sinuses. There is reason to believe that the sinusitis has an etiological relationship with the associated bronchitis. Relief of the sinusitis is usually followed by diminution in the bronchitis and asthma, unless the bronchial lesions are old and extensive or associated with myocarditis. Operative treatment of the sinusitis consists in adequate removal of diseased conditions in the ethmoid bone with the establishment of large openings into the antra to permit the spontaneous evacuation of the antral fluid.

LII.

BARANY'S PRIORITY.

By Irving Wilson Voorhees, M. D.,

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In the Acta Laryngologica, Vol. III, Fasc. 4, which has just come to hand (June 1, 1922), the first article is entitled, "Barany und die Wiener Universität." Five gentlemen, namely, Burger of Amsterdam, Forselles of Helsingfors, Holmgren of Stockholm, Schmiegelow of Kopenhagen and V. Uchermann of Kristiania have taken up the cudgels for Dr. Barany and have set down all the available facts regarding the controversy which has waged about Barany's name ever since the recognition of the prize award by the Nobel Prize Committee. Rumors have come to us on this side that there was a good deal of dissension in Vienna at the time of Dr. Barany's election to the Upsala, Sweden, faculty, but most of it has been hearsay and in the nature of "backstairs gossip," therefore, it seems only fitting to place before the many old students and admirers of our distinguished confrére what this tempest in a teapot is all about.

A short time after Docent Barany had received the Nobel prize for his essay on the Physiologie and Pathologie des Bogengangapparates beim Menschen. (1907), Professor Victor Urbantschitsch recommended Dr. Barany to succeed to the chair of Otology in Vienna University. To the astonishment of those who knew about it, this recommendation was not ratified by the Vienna medical faculty, but no explanation was forthcoming until contumely and canard reached out so far that Dr. Barany was obliged to take recognition of the state of things and ask for an explanation. It then came out that the attack was in secret except for the open attitude of Herr Tandler, Professor of Anatomy. Barany was unable, however, to get any "official" information from the Dean of the University, nor was he allowed to come before the so-called "Senate" to answer such charges as had become the gossip of two continents. He then took the matter higher up, to the Minister of Education, who seems to have succeeded in getting the matter up for discussion. The specific charges accuse Barany of bad faith in claiming originality, and willful carelessness in the use of scientific material. Specifically he had said that certain disturbances in the musculature of the torso are localized not in the hemispheres of the cerebellum, but in the worm. A patient on whom he had made this diagnosis died and he presented the autopsy findings which showed an abscess in the worm as he had said. He failed, however, to mention the fact that there was also an abscess in one of the hemispheres, although the cut brain section showed it, and it was so stated in the published report of the case. The history of this patient with all of the findings had been presented at the congress in London, and later to his confréres in Vienna.

Another charge had to do with "Gross negligence in making false statements as well as attempting to increase his own prestige at the expense of the work of other investigators." In Barany's defense his coadjutors cite two discoveries that are most certainly his: (1) Caloric nystagmus; (2) influence of position of the head upon the falling reaction or direction. The accusers deny the strict interpretation of these statements and say that he took the work of Breuer and Ewald and published it as his own. His defenders say that such work was basic, to be sure, but Barany carried it much further, established it and made it practical, especially as to the facts relating to eye and head nystagmus, about which Breuer said nothing. His defenders say that otology is indebted everlastingly to Barany for the following:

1. That we can produce a caloric reaction in a sound, healthy human ear with warm or cold water.

2. That this reaction is constant and can be definitely described and measured.

3. That he first described "contrary nystagmus" produced by heat or cold.

4. That he first described the changes or reversal in the direction of movement by altering the position of the head 180 degrees.

5. That he first called attention to the dependence of nystagmus upon position of the head in contradistinction to rotational, galvanic or pressure (mechanically produced) nystagmus.

6. That he demonstrated caloric nystagmus to be the result of endolymph movement—a concept which was mentioned by Breuer among other things, but first set down by Barany as the true explanation.

Furthermore, as to the charge that he did not give credit to other investigators there is Politzer's Geschichte der Ohrenheilkunde, in which Barany mentioned in his article the names of Hitzig, Baginsky, Bornhardt and Breuer, merely stating that he, himself, had discovered and applied the laws of caloric nystagmus, not the original facts. In 1906 he published a comprehensive work on the results of his "investigation into the reflex rhythmic nystagmus arising from the vestibular apparatus of the ear and its associated symptoms," in which he acknowledged that the facts came from Purkinje's work, also those of Breuer, published in 1874; but, unfortunately, in his 1909 report before the Budapest Congress he forgot to give credit a second time to Breuer which made it appear that he, himself, had made the discoveries, thus playing directly into the hands of his enemies. This was, indeed, a grievous error which he has tried to acknowledge and to apologize for, but which will not down.

Out of the maze of vituperation and invective the character of Breuer looms up large, and he absolves Barany from all and every alleged attempt to deceive. In a letter dated April 30th, 1921, he says: "I have found two reprints of my first labyrinth work, published in 1874, and send herewith one to you and one to Hofrat Meyer. You will see that I had not then observed and had not described the falling reaction resulting from change of head position. I cannot accept priority of this nor of the caloric reaction in order to injure Barany. I would not do so even if I had cause to complain of any grievance. So much the better is it that my refusal to claim priority rests upon the supreme truth. Therefore, if you are interested, convince yourself of the veracity of my statement—if not, throw the reprint into the fire or return it to me."

There are other statements from the five learned gentlemen which go to prove that the accusations do a great injustice to Barany, but enough has been cited, perhaps, for the American reader to draw his own conclusions. In closing the five professors say: "We honor Barany as the first man since the days of the great pioneers in otology who has opened up a new epoch in clinical experience, and we protest against the attempts of his hidden enemies who would belittle the significance of his scientific contributions and would besmirch his honor and integrity."

SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of February 6, 1922.

THE PRESIDENT, DR. ROBERT SONNENSCHEIN, IN THE CHAIR.

PRESENTATION OF CASES.

DR. H. C. BALLENGER presented a patient with two

Canine Teeth Which Had Extended Up Into the Septum.

She had come in complaining of nasal discharge and crusting and these teeth were found incidentally. She gave a history of all the first teeth having to be pulled with difficulty. Although the girl was twelve years old, all of the upper molars were still the first teeth. The Wassermann reaction was positive. Apparently of the hereditary type.

DR. JOHN A. CAVANAUGH presented an

Instrument for Removing the Anterior Wall of the Sphenoid.

This instrument was an electrically driven burr, which is hooded so that there is only one cutting surface. There is a mark on the handle so that one can tell at all times just where the cutting surface is located. With this instrument the thick lower wall can be removed, and better drainage obtained than can be had by any biting forceps.

DR. G. W. BOOTH presented a

Forceps for Removing Peanuts and Similar Fragile Bodies From the Bronchus.

The instrument was very light and preserved the sense of touch.

DR. NORVAL H. PIERCE addressed the Society on

"A Skiagraphic Study of the Temporal Bone in Relation to Normal and Abnormal Pneumatization."

Dr. Pierce stated that since 1917, when Wittmaack's book appeared in Germany, he knew of no work that has proved or disproved his results. Nearly five years have elapsed and nothing has yet been published that would disprove his conclusions.

Wittmaack divided the pneumatization of the temporal bone into three stages; the first stage covers the first year or thereabouts and consists in the pneumatization of the cavum and the antrum. Up to this time there is no pneumatization of the mastoid process. At the end of the first year and the beginning of the second pneumatization begins in the mastoid, and at the end of the fifth year should be complete under normal conditions. These cells, the concentric and excentric, form the antrum. The third stage lasts through life. As long as a man lives, if there is no arrest in pneumatization due to inflammation which occurs in the first years of life, his mastoid progressively pneumaticizes and this process occurs as it does in the original process, by the invasion of narrow spaces from preexisting pneumatic spaces. This process has been discovered in men as old as seventy.

The pneumatization of bone is really of secondary importance in the process to the changes which take place in the mucous membrane. In order that a pneumatic space may be formed, it is absolutely necessary that the epithelium shall gain entrance to that space. Otherwise, no space can be formed. In many of these spaces there is the narrow space invaded by this submucous myxomatous tissue, but unless the epithelium penetrates into it this myxomatous tissue changes, after it causes the absorption of the marrow proper, into connective tissue, and when this occurs, instead of the marrow spaces being absorbed, there is apposition of this connective tissue and we have the process of sclerosis occurring. In this way the sclerotic bone originates. It is not due to long-continued suppuration but to a failure in normal pneumatization of the mastoid process. Although the red marrow spaces stand at hand ready for the invasion of the myxomatous tissue, nothing of the kind takes place.

What has formerly been called tubotympanal catarrh depends on the arrested pneumatization, and we gain knowledge by Roentgen study of the condition of the mucosa, because when there is arrested pneumatization we know of necessity that there must be a certain type of submucous tissue, because the condition of the submucous tissue is what has arrested the pneumatization. When certain changes have taken place the epithelium is unable to go down into the bone in order to pneumatize it. In all these cases we have a hyperplastic condition.

In the acute inflammation these cases never come to a frank mastoiditis. That is easily understood, because the anatomic parts are not there for the production of this picture; namely, large, well distributed pneumatic spaces.

In the rather arrested pneumatization there is an entirely different picture. We do not find the spaces that are found in the perfectly pneumaticized mastoid. Instead there is a large mass of bone, and if cells are formed those cells have very thick walls, which are very much more resistant to the softening process, naturally, than the very thin, delicate walls of the pneumatic spaces in normally pneumaticized mastoid bone. Not only that, but this hyperplastic tissue is a protection in itself from the bone. In the normal case there is a very thin mucosa, and in the arrested pneumatization a very thick hyperplastic mucosa. In these cases we have the chronic, running ears after this invasion, not a frank mastoiditis. The discharge comes from the tube, from the cavum and from the antrum, but the bone itself seldom softens.

Dr. Pierce disagrees with Wittmaack in that he has found numerous cases of partially arrested pneumatization in which symptoms have developed which threatened the life of the patient because of the invasion of neighboring parts, principally the sinus. These are only in partially arrested pneumatization. In the totally arrested cases it is rare that an involvement is found of either the labyrinth, the cranial cavity or the sigmoid sinus by extension of the softening process, as would occur in very slightly arrested pneumatization or in normal pneumatization. In those cases we do have involvement of the sinuses, but this is by way of the vascular system. The abnormal pneumatization of these vascular remnants between the cells and the blood vessels or meninges occurs and one must give a very careful prognosis in these cases. The vascular connection is probably beyond operative skill.

Dr. Pierce believed these very valuable conclusions could be drawn from skiagraphs. We can tell whether the mucosa is thin. We can tell whether pneumatization is complete. We can tell whether the pneumatization is complete with hyperplastic mucous tissue. We can tell whether there is incomplete pneumatization with a hypertrophic mucous membrane. We can tell whether there is incomplete pneumatization with fibroid mucosa, and all of these have great bearing as to our prognosis and our treatment. For instance, if we have a running ear, without cholesteatoma and with greatly arrested pneumatization, we may know that such a case does not of necessity require operation. That discharge does not come from bone because we know that the bone in these cases is highly resistant. We know that the discharge comes from the highly plastic mucous membrane. We know that in these cases softening is not likely to occur, and if we do operate, the only thing to consider is a radical operation. If we do not do a radical, then we do not drain the cavum, and if we do a radical, then we cannot remove the thickened, hyperplastic tissue of the cavum and the tube. And unless we are quite sure of our technic of closing the mucosa in the cavum we still have an ear that discharges as much as before, and we will have operated on a case that was not dangerous, and will not have improved the patient at all.

DISCUSSION ON PAPER OF DR. NORVAL H. PIERCE.

Dr. Joseph C. Beck said it was a great pleasure to again hear this subject presented by Dr. Pierce. At the time of his previous presentation he was perhaps the only one who was in possession of Wittmaack's book, and in the discussion Dr. Beck mentioned the importance of radiographic study in connection with the work. He now agreed with Dr. Pierce that it should be of the greatest value in connection with the study. He did not believe that Dr. Pierce thought that by means of the X-ray one could determine exactly the type of mucous membrane, but only by inference from the bony changes which were present. In a bilateral, suppurative process it would be difficult to utilize this subject, for when both sides are involved it is difficult to see just what kind of a process is going on.

After receiving the book of Wittmaack's and discussing it with several authorities in this country, Dr. Beck had written and asked Dr. Wittmaack what he would think of having his book translated into English. Dr. Wittmaack was anxious to have this done and had given permission for the plates to be used in this translation, and the work is now being done by Dr. Bigelow of Providence.

Dr. Beck was sure that all saw the importance of the work as brought out by Dr. Pierce.

DR. J. HOLINGER was of the opinion that this explained a number of points that are important in the formation of cholesteatoma. In cholesteatoma there are usually small cells and a small antrum, not larger than a bean. Dr. Holinger did not like the expression, "sclerosed mastoid," but preferred solid or "eburnized." He believed the sclerosed mastoid might be confused with otosclerosis, with which it had nothing to do. He believed that in the cases that used to be called dangerous mastoids, with a thick cortex and a small antrum filled with cholesteatomatous masses and debris, the antrum often became enlarged through absorption, not of inflammatory nature, but purely pressure absorption on account of enlargement of the cholesteatomatous masses. After the operation the horizontal canal was found opened: One would find a tiny groove on the inner wall of the aditus which was nothing but the opened horizontal canal. The process in these cases is always very slow. These points were offered here in explanation of a case shown by Dr. Holinger some time ago, where a man in perfect health suddenly showed protrusion of the posterior superior wall of the canal and a small cholesteatomatous mass was expelled. This opening closed, but after a few weeks another of these masses appeared, and this was repeated five or six times. He now had another patient in whom the same process has been going on for a year. Suction helped to deliver the masses. There was no sign of fresh or old inflammation and no discharge. After that the wound closed and the bulging disappeared. Dr. Holinger thought the explanation given by Wittmaack and Dr. Pierce was much more plausible than the other explanation—that perhaps as a child this patient had a suppuration in the antrum and immigration of the epidermis from the external canal into the antrum, causing displacement of the lining of the antrum and afterwards closing the tympapic membrane over it.

As to the original cause that Wittmaack gives for the whole process of arrested pneumatization—the entrance of amniotic fluid through the eustachian tube into the middle ear during birth with consequent arrested pneumatization and arrested change of the cuboid and cubic epithelium into the normal flat epithelium, Dr. Holinger thought this a little bit far-fetched. In every newborn baby the lungs, the nasopharynx and windpipe are filled with amniotic fluid. The statement that the inflammation caused by that amniotic fluid produces this whole process, Dr. Holinger thought needed confirmation.

Dr. G. W. Boot thought that instead of the chronic, catarrhal ear being the result of the myxomatous tissue it might be the other way around and that because of the air not entering the cavity the submucous tissue was not absorbed.

Dr. Alfred Lewy thought Dr. Pierce was taking a very pessimistic view in saying that a child who gets amniotic fluid into the ear with resulting defective pneumatization of the mastoid was due for a life of ear difficulties. He also removed from the small list of conditions leading to deafness that we have heretofore thought curable, tubotympanic catarrh, so that nothing was left that we can treat successfully but acute inflammatory conditions. Of course, his observations must all be subjected to clinical checking up, like the cases mentioned by Dr. Beck.

Dr. Lewy had recently had under observation what appeared to be a thoroughly pneumatized mastoid, which, according to the X-ray picture, had cells extending into the zygoma and even into the squama, but on operation no cells were found in the zygomatic or squamosa regions, merely diploetic bone, so we must be careful in interpreting our X-ray pictures.

He believed that it would take a great deal of clinical investigation to determine whether the failure of pneumatization was always to result in incurable ear conditions, or was always the underlying cause.

Dr. G. E. Shambaugh called attention to the symmetry usually existing between the mastoid process on the two sides. Where one is small and undeveloped, the other as a rule is small and undeveloped. Where one is large, fully pneumatic, a similar condition exists on the opposite side. Where one shows only a partial pneumatization, exactly the same type of condition is to be expected on the opposite side. For these reasons he was not ready to accept the view that the extent of pneumatization or even its complete absence is necessarily always dependent upon the otitis media. On the other hand, he was willing to accept the view that chronic suppurative otitis media occurring before pneumatization has taken place prevents this from developing. The reason why nonpneumatic sclerosed processes are found in connection with chronic discharging ears is due often to the fact that pneumatization is prevented because of the chronic suppuration. On the other hand, he believed that in acute otitis media with an involvement of the pneumatic spaces, the membrane lining the air cells undergoes inflammatory thickening, leading eventually to the genuine sclerotic process. He has sections of such a mastoid process showing these inflammatory changes.

Dr. Shambaugh also called attention to excessive pneumatization as the direct opposite of the condition discussed above. Pneumatic cells develop at a distance from the confines of the mastoid process. For example, around the tympanic orifice of the eustachian tube internal to the digastric groove and toward the apex of the petrus bone. He has seen cases where the skiograph showed pneumatization spreading back to the torcula.

Dr. John A. Cavanaugh said he had been doing work along the line of otosclerosis and as Dr. Pierce was showing the pictures, Dr. Cavanaugh recalled some points in connection with his own work.

Dr. Cavanaugh thought the marrow spaces which Dr. Pierce pointed out resembled the histologic specimens of otosclerosis. The anterior part, where we recognize the beginning of otosclerosis, he believed, showed an arrested stage. The anterior part of the oval window, according to Huxley, is developed from the opisthotic osseous centers, and this Dr. Cavanaugh thought is arrested in the process of development, and later on, owing to something, whatever it might be, starts the production of bone cells. He has not had any human specimens to work with because they are hard to get, but has been working on young cats, about one month old. Dr. Cavanaugh thought some of the pictures shown were very similar, and that it might have some bearing on the cause of otosclerosis.

Dr. Robert Sonnenschein said that Dr. Pierce's reference to the absence of a frank mastoiditis in the cases of arrested pneumatization was well borne out in a case under observation for several years, a patient Dr. Pierce had also seen several times. This patient was a boy of seven; at four years he developed an otitis media, had another attack at five and another last year. Each attack begins with very mild rhinopharyngitis. A parencentesis is done and a diffust discharge begins. This condition persists for about ten weeks. Ordinarily one would expect a breaking down of the trabeculae but this does not occur. After each onset the patient entirely recovers until the next attack. The pictures taken during the last one showed the arrested pneumatization.

Dr. Sonnenschein thought it was unfortunate that Dr. Pierce could not show all of his X-ray plates and thanked him on behalf of the Society for his excellent presentation and for so extensively studying the subject.

Dr. Pierce (closing the discussion) said that the picture he showed of a man of sixty might well be considered that of a child of a year and a half. There was not a single area in the bone which one could possibly imagine to be a pneumatic space that had been closed up by sclerosis.

He agreed with Dr. Holinger that it was well to keep away from cholesteatoma because this was an entirely different problem. The explanation of Wittmaack was the most intelligent explanation we have at present.

As to the causation of this trouble, the entrance of amniotic fluid into the tube acting as a foreign body, Dr. Pierce thought was the best explanation we have at present, and it is known that several observers who have performed thousands of autopsies on infants have found this condition present. Long before they associated it with arrested pneumatization they found all these histologic changes present, and Wittmaack had said that on account of the small size of the children's clinic at Jena it was difficult for him to find a normal pneumaticized bone—that they were practically all types of arrested pneumatization. All observers have put the process of otitis media neonatorum at from 85 to 90 per cent of infants. Zuckerkandl's description of the three types of mastoid which have hitherto been regarded as normal, the mixed type, the sclerotic type and the rheumatic type, gives statistics which are not far off from the statistics of Wittmaack. The infant mortality must also be considered. Many of them do not grow to manhood. Insurance statistics and vital statistics might clarify the question somewhat as to how many children die in infancy. Dr. Pierce said he was not an advocate; he did not believe that the causation had been proved. As Dr. Lewy said at the last meeting, it might be due to abronmal secretion of the endocrins. The ears are sterile in the great majority of cases and the bacterial infection is due in the great majority of cases, where the inflammatory element is introduced which produced certain changes, to the pneumococcus.

Dr. Shambaugh had called attention to the symemtry of the mastoids, but Dr. Pierce could prove by at least four of his plates that a symmetry occurs with relative frequency. In one case there was partially arrested pneumatization and in the other ear almost totally arrested pneumatization.

As to the entrance of air into the cavum causing this disturbance, this was unthinkable to him. This process takes place in birds as well as in humans and he believed this did not enter into the consideration.

As to Dr. Cavanaugh's trying to mix this proposition up with otospongiosis—not otosclerosis, which Dr. Pierce thought should be stricken out of medical literature—he believed otospongiosis had nothing to do with sclerosis. That is changing the bone into spongy tissue, and Wittmaack has been particularly careful to say that this process has nothing whatever to do with otospongiosis.

Dr. Frank J. Novak, Jr., presented his inaugural thesis, entitled:

"Electrocoagulation in the Treatment of Carcinoma of the Larynx."*

*See page 878.

DISCUSSION.

Dr. A. B. Kauffman emphasized the advantage of diathermy in the treatment of carcinoma in general. In considering the reasons why carcinoma recurs, apart from the factor of stimulation, cutting and removal of sections which often causes rapid growth of the malignant structure, the question of incomplete surgical removal must be considered. One important factor about diathermy is that it acts on the socalled migratory cells and thus reduces the possibility of recurrence. One of the reasons for recurrence of the malignancy is the infiltration of the carcinomatous cells into the adjacent normal tissue, where they are found in the lymph channels and other locations. Dr. Kauffman believed this method was of particular advantage in this respect. Malignant cells are known to be destroyed by heat at about 50° C. and the normal by 60° C. Using this as a working basis one has, in addition to the zone of coagulation, another zone at the periphery where the heat is raised to about 50°, which will inhibit the growth of the cells in the normal tissue. Dr. Kauffman thought the work of Dr. Corbus was confirmed by that of Clark, who suggested preradiation to inhibit the action of the migratory carcinomatous cells, as complementary to diathermy. The depth of heat penetration is found to extend 6 to 8 centimeters beyond the depth of the zone of coagulation, and accounts to a certain extent for the success that can be hoped for in the treatment of carcinomas in general.

Dr. Kauffman thought one of the dangers lay in using too strong a current and there was also the danger of attacking the larger vessels and nerves in the vicinity. In malignancy of the larynx great care must be exercised not to use too great a current on account of the carotid, because it is known that the heat penetration is far beyond the actual contact. One must be very familiar with the technic used and with the amperage which may be used with safety on the different tissues. Fat is very penetrable and the cartilage is more resistant, thus preventing the penetration of heat into the tissues which appear healthy and normal, but which in the lymph tissue and ducts contain the migratory cells which later may become veritable germinal centers for recurrence of the malignant cells.

Dr. Joseph C. Beck thought that anyone who wished to do something for carcinoma in any part of the body could feel encouraged from this presentation. When Nagleschmidt first presented his book Dr. Beck was much interested in the method applied and was glad Dr. Novak had not mentioned his publications in 1907, because he knew now it was not surgical diathermy, but simply charring the parts, as he then understood it. Dr. Beck believed Dr. Novak's work would be of great value and they expected to apply it. He had asked Dr. Orndoff to be present and listen to the paper so that the method could be used in their work. He agreed with Dr. Novak that time will show just what can be accomplished.

Dr. Beck again presented the man who was shown at the January meeting to demonstrate the improvement in the voice with the use of the artificial larynx of Burns. He believed that if a patient lived through a laryngectomy and could use this appliance so that he could talk intelligibly he should be satisfied with the result.

Dr. Alfred Lewy said he had a limited experience with diathermy in carcinoma of the throat, although none of his four cases were in the larynx. He had been afraid to undertake this work in the larynx because of the supposed danger of aspiration of infection from the slough which follows this operation. His method was a little different from that of Dr. Novak. He had used hyoscin, morphin with ether anesthesia, using very little ether, which was always withdrawn for a minute or two before the operating electrode was inserted, and he used a large needle instead of a flat electrode. This had the advantage of requiring less amperage in order to obtain the same results. An area of about one centimeter in diameter around the needle is coagulated in perhaps two or three seconds instead of twenty, so the question of vapor obscuring the sight hardly arises.

The relief from pain in one case was marvelous. The patient had hardly been able to swallow even liquids for about a month, but the day following the operation he was able to eat a square meal. The three other cases were practically not relieved until after the slough separated.

Dr. Lewy believed the fear of hemorrhage was not altogether justified. His cases had been in the region of the tonsils, and side of the tongue, and he had to work rather close to the large vessels of the neck. He had been perhaps rather bold, but he believed that under the circumstances in dealing with malignancy which offered no hope unless completely destroyed we were justified in taking this risk, as death by hemorrhage was perhaps to be preferred to death by cancer. However, there was no such trouble in any of his cases.

In January this year he had shown a patient who was operated some sixteen months before and was in good health with no sign of recurrence or metastasis. Another patient had no recurrence in situ, but died of metastasis after nine months of complete relief so far as the throat was concerned. Of the other two one had been extensively rayed before trying the coagulation but without effect. The other, who had a growth involving the side of the tongue and floor of the mouth, went on from bad to worse.

Dr. Lewy knew of four machines on the market; probably there were more, but he did not know the advantages of each, except that the newer ones were capable of much higher amperage and better control. He believed this method would prove a valuable addition to our armamentarium in the treatment of carcinoma, especially of the mucous membranes. He asked Dr. Novak how far back his cases dated, and whether or not aspiration pneumonia had occurred in any of them.

Dr. Samuel Salinger believed that Dr. Novak deserved much commendation for taking an interest in these cases, most of which were absolutely beyond the hope of surgical intervention. He had seen several of them at the County Hospital and thought Dr. Novak was very modest in withholding his results obtained so far. He knew of two cases in which the progress of the growth was arrested, the patients were much more comfortable and had begun to take on weight. Dr. Salinger thought this particular method of attacking malignant growths in the larynx was more applicable to the advanced cases than the early ones. He still held to surgical intervention in the cases that are intrinsic. He believed there was a great danger of hemorrhage from diathermy in the pharynx and knew of one case that came to a fatal termination shortly after treatment. Whether the diathermy was directly the cause, or whether the malignancy had already reached the large vessels, he could not say.

Dr. Norval H. Pierce asked what the probabilities were of stricture of the larynx developing. If it was true that diathermy caused the production of connective tissue, was there not danger of stenosis occurring even without stopping the growth of the carcinoma?

Dr. Frank Novak, Jr., (closing the discussion) said that the cautery was an accepted method but its limitation lies in the fact that its heat penetration is very slight. The high frequency current has not these limitations but has great penetration.

As to Dr. Lewy's fear of attacking the larynx because of the danger of aspiration pneumonia, so far no case had developed an aspiration pneumonia. He had three cases of carcinoma of the tonsil, four of the tongue and four of the larynx, and one of the palate. Regarding the use of the needle instead of the electrode he used, there is a greater possibility of carbonization with the needle, with a resulting interference with the penetration of heat. His cases had been under observation for about six months.

Dr. Novak did not agree with Dr. Salinger that the method was more applicable to the late than the early cases, because the late cases of carcinoma are hopeless from any standpoint. Metastases always have occurred and treating the original site of the carcinoma only prolongs the patient's life. In his opinion the most suitable case is the early one, where the growth is circumscribed and limited and any invasion of the lymphatic tissue in the vicinity of the tumor by the migratory cells can be coped with by the deep penetration of heat.

Replying to Dr. Pierce, he thought his remarks were interesting, but so far he had not seen any stricture. The scar that is formed is smooth and if one can go by the results obtained by the urologist in work in the bladder, the possibility of stricture is remote. They have found that they can use this method in bladder in the immediate vicinity of the ureters without causing any stricture. The opening persists and no stricture results. He thought it would be possible to tell more in this regard at a later date.

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LIII.

MISHAPS IN THE PUNCTURE AND IRRIGATION OF THE MAXILLARY SINUS.

BY W. E. GROVE, M. D.,

MILWAUKEE.

In any diagnostic measure which we employ, the patient has the right to ask of us, nay, more, to demand of us, that it shall not be fraught with any danger to himself. And if, furthermore, we use this same measure as a therapeutic procedure, the injunction to make it safe to the patient is no less binding upon us. Is the puncture and irrigation of the maxillary sinus as now practiced by the majority of rhinologists perfectly safe and harmless to the patient? If it is not, in what does the lack of safety consist and what can we do to remedy the situation? These are the questions I am asking myself in this paper and which I hope to be able to answer satisfactorily.

HISTORICAL—SOUNDING AND IRRIGATION FROM THE NATURAL OPENING.

As early as the year 1761 a French dentist, Jourdain, was able, by sense of touch alone and without using any artificial illumination whatever, to pass a sound through the ostium maxillare into the antrum. By irrigating the antrum through this route he was enabled to cure many patients suffering from maxillary sinus infection. The procedure, however, soon fell into disrepute and we hear nothing more about irrigating the antrum until 1883, when Hartmann reported three cases cured by means of a dull bent canula. He stated that he began using this method as early as 1874. By 1889 he had collected 32 cases and by this time he had made the discovery that, if he could not readily probe the ostium maxillare or accessorium, he could easily push his dull canula through the posterior fontanelle.

PUNCTURE AND IRRIGATION FROM THE MIDDLE MEATUS.

Thus it is seen that, although Hartmann cannot claim to have been the first to irrigate the maxillary sinus from the nose, he was, in fact, the first to irrigate this cavity through an artificial opening, which he produced by pushing his dull canula through the posterior fontanelle of the middle meatus.

Hartmann's dull canula soon gave way to the sharp canulæ, needles and trocars under the tutelage of Gerber, Killian, Siebenmann, Denker, Körner, Oppikofer, Krebs and others. In fact, Zuckerkandl, writing from a purely anatomic standpoint, gives this reason as a spot of predilection for entry into the antrum because of its ease of access and the thinness of the posterior fontanelle. Oppikofer states that the puncture can be much more easily effected from the middle than from the inferior meatus and with much less pain in the majority of the cases.

PUNCTURE AND IRRIGATION FROM THE INFERIOR MEATUS.

This most common method of puncture was first used and described by Moritz Schmidt in 1888. He only made use of a Pravatz syringe with a needle attached for purposes of aspiration. The first actual syringing through the inferior meatus was done by Lichtwitz in Bordeaux in 1890, and the first inflation with air was practiced by Capdepont in 1894. In the use of this method of puncture and irrigation, Lichtwitz admitted failure six or eight times in 200 cases and Hajek only twice in many thousands of cases.

ANATOMIC CONSIDERATIONS.

In his textbook Hajek, drawing largely on the deductions of Zuckerkandl, calls attention to the fact that while the pars membranacea of the middle meatus near the maxillary ostium, that portion usually known as the posterior fontanelle, is the thinnest part of the nasal wall of the sinus, and therefore the portion most easily perforated by needles and trocars, it is, nevertheless, not the place of predilection for a puncture because of the anomaly, so often present, of fusion of this portion of the nasal wall with the orbital wall of the sinus, and the danger, therefore, of entering the orbit during the puncture. This fusion may take place either because of a very low orbital wall or a marked bowing out of the lateral wall of the middle meatus.

Due to inefficient absorption of the spongy bone of the sinus, the nasal wall in the region of the inferior meatus may be of unexpected thickness and present great difficulty to the puncture needle. In fact, in some of these small antra the floor of the antrum may be considerably above the level of the floor of the nose, and Onodi points out that the wall of the inferior meatus may be as much as three mm. in thickness in its lower portion.

Hajek also draws attention to the fact that if the lower portion of the antrum is very much narrowed, either because of a sinking in of the canine fossa or of a bowing out of the lateral wall of the inferior meatus or both, the puncture needle can easily wound the lateral wall of the antrum or indeed completely pass through it into the canine fossa.

Both Zuckerkandl and Gerber have called attention to the occasional dehiscences which may exist in the orbital wall of the maxillary sinus, particularly in the aged.

A very interesting anatomic finding by Gürlitz will have an important bearing in the clinical portion of this paper. He discovered that the veins of the antral mucosa are very numerous. In 58 per cent of his cases in the well injected specimens he found a thick venous plexus, sometimes most marked on the orbital wall, at others on the nasal wall. Whenever this plexus is present, therefore, it is to be found on those walls of the sinus which may be touched or injured by the puncturing needle, whether this enters from the middle or the inferior meatus. When we remember that Gürlitz's work was done on normal antra and when we consider that the mucosa of the infected antrum may be very greatly thickened and polypoid, we see that the veins in this region may reach considerable size.

METHODS OF PUNCTURE.

As I stated in my historical resumé, there are in the main two methods of puncture and irrigation of the maxillary sinus. The method of Cowper, of irrigating from the alveolus, I will not consider at all, because it has largely fallen into disrepute.

Hajek uses two methods of irrigating the antrum seriatim, first probing and irrigating through the normal ostium, and second, irrigation through an artificial opening made into the antrum.

For irrigation through the normal opening he uses a Hartmann canula, which he precedes with a silver probe so that he can bend his canula accordingly.

Puncture and irrigation through the inferior meatus is done only if probing and irrigation through the natural opening is not possible. For this purpose he makes use of a Lichtwitz needle. Entry is made beneath the inferior turbinate through the upper portion of the lateral wall of the inferior meatus close to the attachment of the inferior turbinate and about three cm. from its anterior end. If the puncture is successful the shank of the needle lies tightly against the septum and is freely movable on a fulcrum at the nasal wall of the antrum. Before irrigating he inflates with air, and if the needle point is within the maxillary sinus a peculiar gurgling sound of air entering the sinus is heard. He always uses a Pravatz syringe to determine whether any serous fluid is within the antrum. If the nasal wall is too thick at the spot selected, he tries a point further forward or further back.

For punctures through the middle meatus the point is selected far back in the middle meatus at the position of the ostium maxillare or ostium accessorium. Either dull canulæ or sharp bent needles are employed. Because of the thin consistency of the antral wall in this region, it being largely membranaceous and devoid of bone lamellæ in some instances, very little force is necessary, and therefore very little pain is occasioned the patient. Hajek warns against the danger of entering the orbit at this point because of the frequent outward bowing of the meatal wall and its frequent fusion with orbital walls. Boenninghaus, on the other hand, does not believe that injury to the hard orbital floor is possible, particularly as very little force is necessary, and if one goes into the hindermost portion of the fontanelle, thus as far as possible from the orbital floor.

Hirsch believes that we should not suit our own convenience in these cases but do the thing which is most comfortable for and least damaging to the patient. For this reason we should select the puncture from the middle meatus, which is far easier and less painful than puncture through the inferior meatus and "here one can push through the fontanelle with the least possible danger."

Heindl draws attention to the fact that considerable force is sometimes necessary to puncture a thick inferior meatus wall and that if the needle is pointed too high and force used to push it through, the orbit can easily be entered with disastrous results. On this ground he advises the middle meatus route in those cases where the wall of the inferior meatus is unduly thick. According to Kelley, Zarniko and Onodi, the antrum should only be entered from the middle meatus in children up to the age of the eruption of the first permanent molar because previous to this age the floor of the maxillary sinus is too high to warrant an inferior meatus puncture.

VALUE OF THE PROCEDURE.

The opinions concerning the value of the puncture of the antrum seem to be greatly divided in recent years. In spite of the accidents which he reports which occurred in many thousands of punctures, Hajek regards puncture and irrigation of the maxillary sinus as a harmless procedure. He warns, however, against believing that one negative puncture spells a healthy sinus. In some of his cases he punctured repeatedly and at various times during the day.

Skillern says that puncture and irrigation should always be tried first, because it is surprising how frequently even the severe forms of the disease recover under such simple procedures. Furthermore, writing in 1913, he states that he had never seen a single untoward symptom in his experience of perhaps 1,000 needle punctures. I think, however, that before we finish this paper we must take exception to his statement, "the needle puncture through the inferior meatus offers the safest, surest and easiest method of ascertaining the contents of the maxillary sinus."

Dutrow, in his article, says: "However, in passing, I wish to condemn the promiscuous use of the trocar and irrigation as a diagnostic routine." "This procedure is not without danger to the patient, as reported by Gording in an excellent paper"; "in which he reviews nine cases found in a literature, with a fatal issue in four, and seven cases collected by himself with two fatalities. I cannot see why we should subject our patients to this added risk when the findings are of so little value." "The puncture and irrigation, to my mind, is devoid of any curative value whatever."

In the discussion of Dutrow's paper, J. F. Barnhill says: "I have yet to see a death or even dangerous symptoms following this valuable diagnostic measure."

Onodi finds irrigation from the middle meatus of enormous value, not only in the treatment of acute cases but in the chronic cases as well.

Because of such serious results as collapse, temporary blindness and even death after forcing air into the antrum of Highmore, Caesar Hirsch has entirely given up the procedure and declares its use unnecessary.

H. W. Loeb says: "The curative value of this method of operating is not great, except in acute cases."

CASE REPORTS FROM THE LITERATURE.

Case No. 1.—Neuenborn reports, from the practice of a colleague, a case of instant death during the puncture and irrigation of an antrum No autopsy reported. Mode of puncture not given.

Case No. 2.—Henry C. Henrici, as cited by Dabney and Gording, reports a case in which polyps were removed from the nose with subsequent irrigation of the frontal and maxillary sinuses. While one antrum was being syringed, the patient suddenly collapsed, and had an epileptiform attack, with cyanosis and trismus. The patient recovered. On the following day the puncture was repeated with the same effect. The trismus was so severe that the mouth could not be opened with gag. There was spasmodic contraction of the muscles of respiration. Even after a tracheotomy it was impossible to carry on artificial respiration and the patient died. The autopsy findings were negative. The puncture was done from the middle meatus. Henrici offers no explanation.

Case No. 3.—Hajek reports the case of an old man who was both diabetic and arteriosclerotic. There was immediate collapse with the development of a left hemiplegia. Death occurred in 36 hours from cerebral apoplexy according to Hajek, although no autopsy was reported. Puncture was done from the inferior meatus.

Case No. 4.—Kelley, as cited by Dabney and Boenninghaus, reports a case in which he tried to force air through the needle. The patient complained of a tickling sensation in the larynx. He was unconscious for three hours. He then had pain in one arm and one leg and had twitching of the leg. Death occurred in fourteen hours. Route of puncture not noted. No autopsy.

Case No. 5.—In a second case, Kelley had removed polyps and washed out the antrum several times by the alveolar route. At this treatment he merely inflated the cavity with air. The patient became unconscious and died in 24 hours. Route of puncture not noted. No autopsy.

Case No. 6.—Claus reports the case of a young woman who had been twice previously syringed without trouble. Immediately after the third syringing sudden cyanosis with unconsciousness, uncountable pulse, breathing stertorous, pupils fixed. Death occurred shortly afterward. The anesthetic used consisted of 6 drops of a 10% novocain solution and 3 drops of adrenalin solution in the course of 20 minutes. Autopsy findings were negative. Claus attributed the death to heart failure from novocain poisoning, but we must not forget that the patient had twice before been subjected to the same procedure with the same anesthetic without untoward symptoms. Puncture done from inferior meatus.

Case No. 7.—Claus reports a young woman who had been anesthetized by a tampon of 10% novocain and 1% (1-1000) adrenalin to the lower meatus followed by a swabbing with 10% cocain. Puncture was done with the Lichtwitz needle without difficulty and air injected into the antrum. There was immediate loss of consciousness with suspended pulse and respiration. Death occurred soon after. The postmortem showed a hemorrhagic infiltration of the epicardium of the right ventricle. The infiltration could be traced through the wall to the endocardium. There was similar infiltration in the septum-membranaceum. Also slight hemorrhages into the gray matter of the brain, in the aborvitæ of the fourth ventricle and in the lower part of the cerebellum. Puncture done from the inferior meatus. Claus also regarded this as a case of novocain poisoning.

Case No. 8.—Brown reports the case of a male aged 24 in whom the inferior meati were anesthetized with 20% cocain. The right antrum was washed out with negative results. The left antrum was punctured in the same way, but as soon as air was forced in the patient became rigid with backward rolled eyeballs. Then relaxation set it together with profound cyanosis and muscular twitchings. He was placed in the recumbent position. There were very few signs of life. Artificial respiration, whiskey, strychnin and adrenalin used. At the end of 20 minutes the cyanosis cleared somewhat but death occurred one hour after the puntcure. At autopsy when the pulmonary artery was incised a sharp hissing sound of escaping air was heard and the greatly distended heart collapsed. When the antrum was opened the mucosa of the roof, inner and outer walls, was found detached and that portion hanging from the roof was found to have a puncture wound in it while the underlying bone was found to be uninjured. The assumption, therefore, occurred to Bowen that the needle entered the mucosa of the roof and that air was forced between the mucosa and the underlying bone, opening up numerous capillaries. Bowen further notes that very little force was used in making this inflation. Puncture done from lower meatus.

Case No. 9.—Killian reports a case from his clinic which died immediately after irrigation of the antrum. He states that the water used in irrigating was too cold and believes that the death was due to a vague reflex. No autopsy. Puncture done from middle meatus.

Case No. 10.—Gording reports a farmer of 61 in whom the left lower meatus was cocainized with 20% cocain. Perforation in the usual way. This antrum was washed out. Then the right antrum was prepared and washed out. Pain was complained of. There was no dilated pupil or other sign of cocain poisoning. Air was blown through the sinus on several compressions. At last a mucopurulent mass was blown through the ostium which blocked while going through. At that instant patient became cyanosed and respiration ceased. Artificial respiration was done for one-half hour without result. Death. Autopsy not available. Puncture through inferior meatus.

Case No. 11.—This case of Fleischer of Kristiania reported by Gording was that of a young woman. Puncture was made through thick bone in inferior meatus and was difficult. During inflation patient sank unconscious with irregular and stertorous breathing. Comatose condition continued until death occurred, 12 hours later. Autopsy findings were negative. Puncture done from inferior meatus.

Case No. 12.—Schneider reports a female aged 24 who complained of nasal obstruction. Polyps in the nose were removed by snare and conchotome under 10% cocain. Puncture was done from the middle meatus under 10% cocain with a Killian needle. Immediately after beginning irrigation patient collapsed with a barely palpable pulse, and superficial respiration. Death occurred in several minutes. The autopsy showed pus in the antrum, sclerosis of the blood vessels, much serous fluid in the auricles and signs of stasis in the liver, spleen and kidneys. Schneider gives shock as the cause of death. The puncture was from the middle meatus.

Case No. 13.—Neugebauer reports a male of 26 who had a cold for 6 weeks. The right cheek was swollen at the onset. Later pus appeared in the right nose. There were occasional headaches. Nov. 19, 1916, 5:30 p. m., puncture of the right antrum was done from the inferior meatus after cocainization. The needle entered easily. Its point was freely movable. The shaft of the needle was against the septum. The sound of air in the sinus upon inflation was typical. On the second compression of the bulb this sound was no longer heard. Immediate unconsciousness. Transient convulsions. Pulse of good quality, but slow and irregular. The face was flushed and cyanotic. There was no swelling or emphysema of the cheek. After a half hour, consciousness slowly returned and the patient was able to talk. One hour after the puncture Neugebauer left the patient, because he regarded the danger as over, but shortly after this the patient suddenly became worse and died. Autopsy seventeen hours later showed air bubbles like pearl chains in the pial veins of the right hemisphere. Larger air bubbles in the jugular vein. Air in the veins of the visceral pericardium. The heart contained only air and no blood. Foamy blood in the liver on cutting the same with a knife. The antrum was opened from the canine fossa and found filled with much thickened mucosa, polypoid masses and much pus. Puncture was done from the inferior meatus.

Case No. 14.—Kubo reports a case of syncope and death on the day following puncture. Details are wanting. No autopsy report. Route of puncture unknown.

Case No. 15.—Henry Horn cites the case of the patient of a colleague in which collapse and death followed immediately upon drying the cavity. Details are wanting. No mention of the route of puncture.

When we review critically this series of fifteen deaths we find that they have many things in common. In the first place, they may all be classed as sudden deaths, the time between puncture and death being only a few minutes in eleven of the cases and from a few hours to 36 hours in the other four. The antemortem symptoms also in those cases where sufficient details were given were much the same in all of them. There was sudden collapse with unconsciousness, cyanosis, pulse and respiration changes, tonic and clonic contractions of various groups of muscles, or indeed of all the muscles, sometimes designated as epileptiform attacks. In some of the cases muscular twitchings were noted. We must, therefore, assume, from the more or less common symptomatology preceding death in all cases, that we are dealing with a causative factor which is common to all of them. There is nothing in the method of puncture which will assist us, for we find that the puncture was done seven times from the inferior meatus, three times from the middle meatus, while in five cases the route was not mentioned. In the matter of autopsies we find that none was done in eight cases. In three of the cases the autopsy findings were entirely negative. In two of the cases minute hemorrhages or signs of stasis were found in various organs, and in two of the cases air was found in the circulation. What bearing these autopsy findings may have on the solution of the problem I will discuss after the citation of the remainder of my cases. We will also especially bear in mind the very interesting finding in Bowen's case of the detached thickened mucosa of the sinus, in which a needle wound was discovered.

One reported case of death after a puncture of the antrum has not been included in the above mentioned series, inasmuch as it seemed to me that it was very questionable whether the death could in any way be attributed to the puncture. It is given here, however, because it is included by Dabney in his list of deaths due to intranasal operations and other instrumentation.

Case No. 16.—Culbert reports a female patient who had a right sided frontal and maxillary sinus infection following an influenza. Fourteen days after the antrum had been opened by a trocar she awoke with secretion in the eyes. On the following day there appeared a red, itching, papular eruption on the face, breasts, abdomen and arms. There were desquamating areas on the buccal mucosa. The conjunctivæ were injected, giving off a mucopurulent secretion. In the afternoon the eruption became vesicular. This later broke down into foul smelling ulcers. There was an abundant discharge from the mouth, of a sanguinous, mucopurulent character. Later there appeared the same sort of a discharge from the anus and vagina. Constant fever set in, together with vomiting. The patient went into delirium, unconsciousness and died. The diagnosis was that of an acute pemphigus.

It seems to me, in as much as none of the symptoms of the pemphigus in this case appeared until fourteen days after the antrum puncture, the death cannot be justly attributed to the operative procedure.

We will next pass on to the consideration of a second group of cases in which there occurred collapse and serious general symptoms, including suspended or altered breathing, pulse changes, tonic and clonic contractions of muscles, hemiplegias, etc., in other words, the same general symptoms which occurred in group I, with the exception that the patients luckily escaped with their lives. In this group we find twenty-one cases in all.

Cases No. 17 and No. 18.—Hajek reports two cases of unconsciousness lasting several hours and initiated with convulsions. Puncture through inferior meatus.

Case No. 19.—Claus reports the case of a young girl of 19, who had twice previously been treated for left antrum infection. At both times a fairly large opening had been made into the inferior meatus. Two years later she had another recurrence. Only a small fistula remained sufficiently large for the passage of a probe. A new puncture was made with a Lichtwitz needle, which was very difficult because of greatly thickened bone. Air was heard in the nose when the antrum was inflated. Suddenly the sound of the air ceased, and on a stronger compression of the bulb the patient suddenly became cyanosed, was dyspneic, had a feeling of fear but a strong quiet pulse of 75. The feeling of oppression remained for about two hours. Claus himself believed this to be a case of air embolism. The puncture was from the inferior meatus.

Case No. 20.—Claus reports a male of 68 who suddenly became unconscious after a puncture. There was paralysis of the right arm and leg but not the face. The pulse was normal. On the following day the patient was clearer, but his speech was indistinct. There was gradual improvement, and after six days no paralytic symptoms remained. Puncture was from the inferior meatus.

Case No. 21.—After irrigating an antrum Streit noted temporary paralysis of both legs and transient disturbances of vision during which the patient had alternating periods of blindness and vision for over two hours. An ophthalmoscopic examination made by Brücker did not reveal anything abnormal in the eye ground and he made a diagnosis of air embolism in the occipital lobe. Puncture was done from the middle meatus.

Case No. 22.—Neuenborn reports a young man with a double antrum trouble. The left side was punctured and syringed twice without trouble. Two days later the right antrum was punctured with the evacuation of pus and introduction of air. There ensued immediate unconsciousness with cessation of respiration, fixed and dilated pupils, cyanosis and pallor. The entire body was rigid and in a state of spasm. The diaphragm was massaged and after twenty-five minutes a spontaneous respiration began. The spastic condition did not clear up for two hours. The patient was unconscious for the next three days, but with a normal pulse and temperature. Consciousness returned on the fourth day. Neuenborn believed this due to cocain.

Case No. 23.—Henrici removed polyps from a case under 10 per cent cocain. After their removal the antrum was to be syringed through the middle meatus. As soon as the syringing began, the patient had an epileptiform attack with labored breathing, cyanosed face, clonic contractions in arms and legs. The pulse was suspended. Under artificial respiration and heart massage the patient rapidly improved, but for a time both arms and legs were paralytic. There was ultimate recovery, but for two years there were periods of listlessness and forgetfulness. Puncture from the middle meatus.

Case No. 24.—Zarniko punctured through the inferior meatus in a woman of 50. The puncture was done with great ease. When after injection of air Zarniko started to irrigate, the patient suddenly collapsed, with face pale, cold and clammy skin, fixed and dilated pupils, suspended pulse and respiration. There was a gradual return to normal and in one hour the patient was able to go home. Puncture from inferior meatus. Zarniko thought this was due to trigeminal irrigation.

Case No. 25. Gording reports the case of a male aged 54 in whom a diagnostic puncture of the left antrum under 10-20% cocain was done. The bone was very thick and the trocar was forced through with difficulty. The air seemed to meet with resistance. The patient dropped unconscious with fixed but not greatly dilated pupils. There was a cessation of respiration, uncountable pulse. The entire muscle system was in a condition of tetanic contraction. In spite of the trismus the mouth was forced open and artificial respiration begun. After a time spontaneous respirations began. After camphor caffein injections the pulse again returned. Patient regained consciousness after about 30 minutes, but was confused and badly orientated. There were no convulsive movements in this case, but the patient complained of numbness of the hands and feet. In one hour he was able to move his hands and feet. He stayed in bed for two days and could then use his right leg with a limp but not the left leg for two days more. The numbness in the left leg lasted six to eight weeks and was still slightly present in the left foot three years later. Puncture from the inferior meatus.

Case No. 26.—Gording reports a male of 51 who had been for some time under treatment for an acute antrum trouble. The left side had frequently been punctured and irrigated without bad effects. Puncture was then done on the right side through the inferior meatus. The air met with resistance. The patient suddenly became unconscious and cyanosed. Respiration ceased in the inspiratory position. All the muscles of the body were in tetanic contraction. The pupils were fixed but not dilated. The rigidity soon passed into tonic contractions. Artificial respiration begun and camphor caffein injections given. Consciousness returned in 20 minutes. For several weeks he was tired and not interested in his work. Thirty years before, this man had had two epileptiform attacks but no recurrences. Puncture from inferior meatus.

Case No. 27.—Gording reports a farmer, aged 64, with left sided antrum trouble. Puncture done through the inferior meatus, after cocainizing with 20% cocain. No inconvenience from the cocain and no dilated pupils. Careful injection of air showed that it met with considerable resistance. As soon as the pressure was raised the patient complained of difficulty in breathing. Cyanosis, cold sweats and uneasiness set in. He was placed in a recumbent position and the trocar removed. The respiration was shallow and irregular. Pulse was small and hardly perceptible. In 10 minutes the symptoms were better and in a half hour he was able to go home. Puncture through inferior meatus.

Case No. 28.—Holmgren (cited by Gording) reports a female, aged 65, with chronic infection with fetid secretion. Irrigating done on several occasions through a Lichtwitz needle and the lower meatus. Air was inflated before irrigating. The patient immediately collapsed, pale and pulseless. At the same time a peculiar gurgling sound was heard as of air bubbles. They gave the impression of moving quickly down the jugular vein. The patient was exhausted for several hours but then grew gradually better and was completely recovered by the next day. Puncture was done through inferior meatus.

Case No. 29.—Roll Hansen of Kristiania (cited by Gording) reports the case of a young woman always well except for obstructed right side of the nose. The right side was found filled with polyps which seemed to spring up from the anterior ethmoidal region. After cocainizing with 15% cocain some of the cells were removed in the usual way. The patient was quite well after a brief treatment. Later a puncture through the inferior meatus was done. There was great difficulty in passing through the bone because of its hardness. As the needle pierced the bone the patient turned pale. The perforation was completed and careful inflation begun. At the first compression of the bulb the patient fell to the floor unconscious. There was complete rigidity of all the body muscles. The respiration was rapid and stertorous. The pulse was rapid. The attack lasted five minutes and then consciousness returned. Tonic rigidity but no clonic contractions of the muscles was present. For a time after the attack there was paresis of the right arm. After five hours asphasia was present. Pressure of the right hand was weaker than the left. After five days the asphasia and all the other symptoms were gone. The puncture was from the inferior meatus.

Case No. 30.—Kayser (cited by Boenninghaus) reported a hysteroepileptic attack lasting 36 hours.

Case No. 31.—L. K. Guggenheim of St. Louis reports a case of collapse similar to those reported by Gording. He thought the patient would die, but fortunately he recovered. Meatus not noted.

Cases No. 32, 33 and 34.—C. F. Pfingsten reports three cases of near death among his colleagues in St. Louis. Meatus not noted.

Case No. 35.—Kohlhaus (cited by C. Hirsch) reports a case of collapse about one hour or so after irrigation of an antrum with hydrogen peroxid solution. The case recovered.

Case No. 36.—Bowen reports a female in whom he punctured through the inferior meatus after cocainizing with 20% cocain. The puncture was difficult because of unusual thickness of the bone. It was impossible to force air through, so the needle was slightly withdrawn. The second attempt was also unsuccessful. During the third attempt the patient became rigid and cyanotic. A moment later there were convulsive movements of the extremities, frothing at the mouth, and stertorous breathing. She was admitted to the hospital and was unconscious for 72 hours. Not rational for five days. Temperature 101 2/5, pulse 75, respirations 20, on admission. Profuse perspiration, especially at night, and muscle twitchings. On the fourth day involuntary micturition and paralysis of the left arm became apparent. Patient was discharged from the hospital on the seventh day. Puncture from the inferior meatus.

Case No. 37.—Moritz Schmidt reports a complete left sided paralysis of arm and leg together with collapse. In this case the condition followed immediately after the inflation with air of a large posterior ethmoid cell. (While this is not strictly an antrum puncture case, it is reported here because the typical symptoms noted in those cases appeared here also when another bone cavity was inflated with air. This same notation holds in the following cases of Kümmel.)

Case No. 38.—Kümmel noted a transitory hemiplegia after air inflation of the frontal sinus.

Cases No. 39 and 40.—Kümmel reported two cases of long continued unconsciousness without any other symptoms after inflation of the frontal sinus with air.

Case No. 41.—Kümmel reports a comatose condition after antrum puncture. Meatus not noted.

Case No. 42.—H. Horn reports a case with faintness and collapse after puncture and irrigation from the inferior meatus.

This series of 29 cases in all of which the symptoms were very similar really consists of 25 cases of puncture and irrigation of the maxillary sinus and four cases of inflation of other sinuses with air. In these 25 cases the puncture was done 15 times through the inferior meatus, twice through the middle meatus, and in eight cases the route of the puncture was not noted.

Since writing this paper two more cases of collapse during air inflation have come to my attention in the practices of two of my colleagues, and one from the literature.

Dr. C. J. Coffey of Milwaukee reports a female, aged 54, in whom, after cocainization of the right inferior meatus with 10 per cent cocain, he punctured the right antrum and irrigated with normal salt solution. No bad after effects. He then repeated the procedure on the other side, with the same method of anesthesia. He met with considerable resistance in attempting to force water into the antrum and caused the patient some pain. At the same time only very little water returned. The patient immediately collapsed, became cyanotic and unconscious with some convulsive movements. Death seemed imminent. Various methods of stimulation were resorted to. It was fully one-half hour before it was deemed safe to remove her from the operating room to her bed.

Dr. Richard Muenzer of Milwaukee reports the case of a young woman of 18, whose left antrum he entered through the inferior meatus with a curved trocar. Air inflation was done, followed by irrigation with normal salt solution. In drying out the cavity with air the patient suddenly collapsed and became dyspneic. There was some slight pain, twitchings of muscles, the patient was not entirely unconscious, and complete recovery ensued in about two hours. A second puncture, which was done a week later, produced the same train of symptoms.

These cases, in my mind, both enter into Group II.

H. L. Lanier reports the case of a Mrs. B., aged 52, with symptoms of an empyema of the antrum of Highmore—that is, left sided antrum empyema of uncertain duration, who was to undergo puncture of the maxillary antrum. Ten per cent cocain solution had been applied to the left lower meatus, the perforation was performed quickly and easily, in the usual way.

The injection of air was proceeded with, the air at first passed through the antrum without resistance; several compressions of the bulb were made in rapid succession, when a mucopurulent mass was blown out through the maxillary ostium. The ostium then seemed to be blocked, immediately the pressure rose in the antrum, and could be felt in the bulb, requiring greater force to compress the bulb. The lump of secretion from the antrum was then forced out through the ostium, when instantly, without any symptoms of cocain intoxication, the patient fell prostrate, cyanosed, respiration was irregular and shallow, the pulse was small, hardly perceptible. The patient was placed in the horizontal position; stimuli and artificial respiration for half an hour seemed to keep the patient alive, but the patient was still unconscious, with stertorous irregular breathing. This condition lasted for three hours; the patient then regained consciousness, but she was much exhausted and almost motionless for fifteen hours, when she grew better and recovered without any ill effects worth mentioning.

The question is whether her symptoms were due to cocain intoxication or to an air embolus. Personally, I believe an air embolus was responsible for the symptoms outlined in this case.

In the next group of cases the common symptom was transitory blindness.

Case No. 21.—This case of Streit's already cited under group II displayed the symptom of blindness alternating with good vision for a period of about two hours with ultimate recovery in which the ophthalmologist Brückner was unable to detect any eyeground changes and made a diagnosis of air embolism in the occipital lobe. Puncture through middle meatus.

Case No. 43.—Caesar Hirsch reports a female, aged 16, with bilateral antrum infection after grippe. He irrigated both sides from the middle meatus with no bad results. On the next day he washed the right antrum until the fluid returned clear. During the subsequent inflation with air the patient suddenly said, "I do not see any more with my right eye." The pupil was immobile but no pain was complained of. An immediate ophthalmoscopic examination revealed a pale disc, greyish retina and normal veins. In the arteries bubbles of air were plainly visible separated from each other by columns of blood slowly moving. After two or three minutes, the air left the arteries, the retina assumed a more normal color and the disc lost its distinct pallor. The activity of the pupil and the sight also returned. Puncture from the middle meatus.

Cases No. 44 and 45.—Hajek reports two cases of transitory blindness in which an ophthalmoscopic examination made after the cessation of symptoms failed to reveal anything abnormal. Puncture done from the inferior meatus.

Case No. 46.—Killian reports a case in which the vision was lost for several hours after irrigation. The pupil remained dilated for several days.

Case No. 47.—Walliczek reports a case similar to that of Streit (Case 21).

Having enumerated in three classifications the cases in the literature which have many things in common and which are apparently not due to infection or faulty technic, I will now pass on to a large group of cases in which the complications were caused by forcing air, irrigating fluid or products of infection into the tissues adjacent to the antrum during the act of puncture or irrigation. These were in the main minor mishaps and no fatality was recorded in this group.

Case No. 48.—Gerber. Swelling of lower eyelid, which formed an abscess after eight days. A defect existed between the antral and orbital wall. Puncture from the middle meatus.

Case No. 49.—Gerber. Swelling of the lower eyelid region, which subsequently abscessed. Puncture from middle meatus.

Case No. 50.—Gerber. Swelling of the upper and lower eyelids. Puncture from the middle meatus.

Case No. 51.—Hajek. Case with a defect in the orbital wall in which every inflation caused emphysema and every irrigation edematous infiltration of the lower lid. Puncture from inferior meatus.

Case No. 52.—Hajek. Emphysema and infiltration of the cheek, at times going on to fever, chillls and ankylostoma of eight to ten days' duration seen very frequently.

Case No. 53.—Hajek. Emphysema of the orbit and lids. (Case of one of Hajek's assistants.) There were no serious consequences.

Case No. 54.—Nager (reported by Boenninghaus). Abscess of the orbit.

Case No. 55.—Gerber. Abscess of the orbit. Puncture from the middle meatus.

Case No. 56.—Boenninghaus. Abscess of the orbit.

Case No. 57.—Kronenberg reports the case of a male, aged 55. Puncture done through the inferior meatus and foul pus evacuated. The puncture was easily done. During the irrigation obstruction was felt and the patient immediately complained of pain in the face. There was mild infiltration in the infraorbital region. Moist compresses ordered. A few hours later rise of temperature and spreading of the infiltration to the molar region and angle of the jaw. On the following day there was a chill and a livid swelling over the whole cheek. Incision through buccal mucosa advised and refused. On the following day there was a spontaneous rupture and discharge of pus through the buccal mucous membrane. The discharge continued for one week. Puncture was through the inferior meatus.

Cases No. 58 and 59.—Moritz Schmidt reports two cases of swelling of the cheek and eyelids. No bad results. Puncture from the middle meatus.

Case No. 60.—B. Fraenkel. Abscess in the cheek.

Case No. 61.—Douglas Beaman. Swelling of the cheek and orbital region.

Case No. 62.—Luc. Orbital abscess.

Case No. 63.—J. Hollinger. Swelling of the upper and lower eyelids. Disappeared after one week. Puncture from middle meatus.

Case No. 64.—H. Horn. Entered the cheek with the production of an enormous phlegmon. Healed on incision. Puncture from middle meatus.

Cases No. 65 and 66.—R. Sonnenschein reports two cases of temporary emphysema of the cheek. Puncture from the inferior meatus.

Cases Nos. 67, 68 and 69.—G. Shambaugh reports three cases of emphysema of the orbit. No serious after results. Puncture done from the middle meatus.

Case No. 70.—R. Skillern reports the case of an assistant where four ounces of sterile water were injected into the orbit. Recovery in about four days. Inferior meatus puncture.

Case No. 71.—R. H. Good. Swelling of the cheek with no serious after results. Inferior meatus puncture.

Case No. 72.—R. H. Good. Reports a case seen in England of swelling of the cheek from a collection of fluid.

Case No. 73.—T. H. Halsted. Edema of the orbit and conjunctivae. Recovery in two or three days. Inferior meatus puncture.

Case No. 74.—Lee Cohen. Emphysema of the nasopharynx and soft palate. Inferior meatus puncture.

Cases No. 75 and 76.—J. Barnhill. In two cases fluid entered the orbit. No serious after results. Inferior meatus puncture.

Case No. 77.—W. Freudenthal reports in the case of an assistant an alarming edema spreading to the eyelids which subsided under ice. Inferior meatus puncture.

There are two cases of infection with chills and fever and metastatic abscesses in other parts of the body. They are:—

Case No. 78.—Hajek reports the case of a woman in whom he punctured the left antrum. After inflation a few drops of pus were expressed. Severe pain was complained of in the antrum. The needle was removed. The next day there was a chill with a temperature of 40° C. Two days thereafter there was again a chill with a high temperature. At the same time a brawny swelling appeared in the left temple. After two more days with chills and fever a large swelling appeared in the left wrist, which immobilized the same. A Caldwell-Luc operation on the antrum showed an extremely narrow lumen completely filled by swollen mucosa. The patient recovered after this and the metastases disappeared.

Case No 79.—Uffenorde reports a severe streptococcic septicemia and pyemia with many metastases and abscesses following puncture and irrigation. In addition to these cited cases Killian, Boenninghaus, Siebenmann, Kronenberg and Walliczek have repeatedly experienced several hours after irrigation rises of temperature sometimes so high as to be accompanied by chills, though as a rule this phenomenon only accompanied the first irrigation. This must be looked upon as an absorption temperature.

There follows now a report of my own case which occurred a number of years ago and with which my special interest in this class of complications began.

R. M. D., February 17; 25 years; clerk. Diagnosis: Acute maxillary sinusitis left. Subacute coryza. Complaint: Cold. Pain in left eye and cheek.

History: About one week ago patient took cold. Thought it was all gone. Yesterday morning the left eye began to redden. Some pain below the left eye.

Examination: Slight septal deviation and marked spur at vomeromaxillary articulation left. Left side mucous membrane reddened and intumescent around swollen border of middle turbinate. There is pus in middle meatus and between middle turbinate and septum. Transillumination shows the left antrum dark. All other sinuses fairly transparent. Pressure over left molars more sensitive than right. Acute blepharitis in left eye. Suction gives a great deal of pus.

Pus is seen coming from both posterior nares, especially on the left side.

Pharynx is reddened and somewhat swollen.

Therapy: Infraction of middle turbinate done.

March 5. For about one week after admission, treatment consisted of application of cocain and adrenalin to middle meatus; suction and aspirin. After subsidence of temperature irrigation of the sinus every day or every second day. These irrigations were of necessity made from the middle meatus because of deviation and crest of septum. Today after irrigation I introduced about 1 cc. of 5% protargol into the sinus. In attempting to introduce a second 1 cc. the injection produced an extrusion of the bulb and a marked subcutaneous swelling of the upper and lower lid with great pain in and around the eye. Distinct crepitation can be felt in both upper and lower lids near inner canthus. Patient sent to the hospital.

March 11. For 24 hours hot applications to eye with pressure. Free catharsis. Sweating packs daily. After 24 hours the dressings changed to ice compresses. Pressure continued. In the last two days swelling has receded so that pupil can be uncovered. Pupil dilated; no reaction to light. No sensation of light. Great loosening and folding of the conjunctiva, both tarsal and conjunctival. The eye grounds were negative except for slight hyperemia. Disc normal.

March 13. Dr. Seaman examined the eye yesterday. Prognosis bad for sight. The eye movements are much better than they have been. The chemosis of the conjunctiva is retreating. The facial subcutaneous swelling greatly decreased. The swelling of the lids has decreased so that patient can open upper lid alone. Today on light application there is a dilation of the pupil. The pupil is smaller than it has been. No changes in the eye grounds. No light sensation.

March 28. The palpebral slit of the injured eye is still less open than that on the other side. No pupillary reflex to light. The indirect reflex is present. Papilla of the injured eye is becoming very pale. Optic atrophy.

April 9. Patient left treatment.

In the explanation of these various complications we have various factors to consider. In 48 to 79 the explanation is fairly easy and had to do with the introduction of air, irrigating fluids or products of infection into the tissues of the cheek, eyelids or orbit adjacent to the maxillary sinus and can therefore be classed as emphysema or infection or both.

In cases 1 to 47 we have a large group, including 15 deaths with symptoms very similar in all where the details were available, and 29 cases where the symptomatology was similar to that of the death cases, but in which the fatal issue did not ensue.

I believe the causative factor of all of these cases to be the same. It must be remembered that none of these complications occurred at the time of the actual puncture itself, but later during the period of air inflation or syringing. Therefore I believe that the puncture itself can be eliminated. Claus believed that his two fatalities were caused by anesthetic poisoning, nevertheless, quite naively, he considers it very strange that it should happen in two irrigation cases and never in the thousands of other anesthetic cases in the clinic. Neuenborn held the same opinion as to the cause of his fatality. Kronenberg would also classify the deaths of Claus and Neuenborn as anesthetic deaths. Writing in 1913 Boenninghaus expressed himself as not believing that these mishaps were occasioned by cocain or novocain poisoning.

It does not seem to me that cocain or novocain poisoning can explain these complications, for we must remember that many of these patients had had previous operative work in the nose done when as much or more cocain had been used as at the time of these punctures and had not suffered any inconvenience therefrom. Moreover, the collapse in these cases was very sudden, and we know that the cocain poisoning case gives us various premonitory symptoms before actually collapsing. Furthermore, if the mishaps were the result of cocain poisoning we would expect that it would come on at various stages of the procedure, either before the puncture or at the time of the puncture or even later, but, as a matter of fact, all of these accidents occurred either directly upon the air inflation or immediately following irrigation.

We have then two other possible explanations for the phenomena, either a nasal reflex of some sort or the production of an air embolism.

Dabney thinks the only rational explanation for the sudden deaths reported by him is a reflex irritation of the vagus caused by stimulation of the branches of the trigeminus which supply the antrum.

Siebenmann considered the attacks of giddiness after irrigation of the antrum as due to a vagus reflex. Killian was also of the opinion that bradycardia, unconsciousness and even several cases of death were due to an intercommunication between the vagus and the trigeminus.

In 1870 Kratchmer, later Langendorf, and in 1883 Holmgren showed that cessation of respiration could be produced by irritation of the nasal terminal fibers of the trigeminal. Knoll, working with curarized rabbits, found that he could produce respiratory cessation by strong electrical stimuli to the supraorbital nerve and also by pressure on this nerve. He assumes not only a connection between these nerves and the respiratory center, but also a direct connection between these nerves and the spinal centers which directly supply the respiratory muscles.

Sandman, Dixon and Brodie and Lazarus came to the conclusion that irritation of the nasal mucous membrane brought on not only a cessation of respiration but a contracture of the musculature of the bronchioles as well. The centrifugal portion of this reflex must be sought for in the vagus, for when this nerve is cut the reflex does not occur, and it can again then be produced by stimulation of the peripheral cut end, according to Lazarus.

In discussing Dutrow's paper, A. H. Andrews of Chicago said: "I think the greatest number of deaths have been caused by putting air into this cavity and having it go where you do not wish it to go."

Gording found while experimenting on rabbits that he could produce changes in respiration all the way down to total cessation by injecting air into their maxillary sinuses, but no changes in the pulse rate were noted. In one of his rabbits, however, in which he obtained the results just stated when he injected air on one side, death occurred when he repeated the experiment on the other antrum, and in this instance he found that an incomplete puncture had been done and the mucosa of the sinus had been stripped from the nasal wall occasioning bleeding. The autopsy in this rabbit showed air emboli in the right heart and in the general circulation. He, therefore, concludes that in the small thick walled antra where the puncture is made slowly and with difficulty, it is possible to tear loose the mucosa from the underlying bone and open up blood vessels. In these cases the mucosa is pushed ahead of the needle and on inflation air is then directly forced into the circulation. In fact, during several Luc-Caldwell operations, after removal of the canine fossa he pushed a needle through the thick bone of the inferior meatus slowly and was able actually to observe that the mucosa was pushed ahead of the needle. He, therefore, assumes that in those cases of his where the bone was thick and the collapse came on immediately after inflation the causative factor was an air embolism. In the other cases where the bony wall was thin and the symptoms did not come on until actual syringing began, he assumes a trigeminal reflex as the cause of the catastrophe.

I do not myself believe that this last point is well taken, for we know that with the syringe in use among most rhinologists very little time is lost between the cessation of the inflation and the beginning of the irrigation, and it might easily take this small interval of time for the air which had entered the circulation during the inflation to reach the coronaries or the arteries of the medulla.

From his own case of demonstrated air embolism of the arteria centralis retinæ (Case No. 43) and from those of Killian (46), Streit (21), Walliczek (47), Bowen (8), Neugebauer (13), Hajek (44 and 45) and Kohlhaus (35), Caesar Hirsch concludes, first, that the cases formerly regarded as due to vagus irritation are in fact caused by air emboli, and second, that these emboli, where visual disturbances exist, are themselves visible by immediate ophthalmoscopic examination and are recognizable by the pallor of the disc, silvery narrow arteries with air bubbles within their lumina, normal veins and a slightly cloudy eyeground.

It is my belief that these sudden deaths numbering fifteen and all or nearly all of the cases with serious symptoms in the second group of twenty-six were due to air embolism caused during the act of air inflation of the antrum. In support of this view we have the case of Holmgren,[28] whose patient collapsed pale and pulseless upon the inflation and who heard at the same time a peculiar gurgling sound as of air bubbles quickly moving down the jugular vein; we have the case of Caesar Hirsch,[43] where the air bubbles were demonstrated during life in the central artery of the retina, and we have the cases of Bowen[8] and Neugebauer,[13] in which the autopsies showed air in the circulation, also the rabbit of Gording's experimental series which died of air embolism.

In addition to these cases, Kelley relates an interesting case which I have not included among my case reports because no deleterious symptoms were experienced, of a woman whom he punctured from the inferior meatus. He believed the canula to be in a good sized antrum. On inflating a low gurgling sound was heard unmistakably passing down the neck. The patient said she felt something passing down the neck but was in no way affected by it. Kelley felt that the needle must have passed through both walls of the antrum and opened up a vessel in the pterygoid plexus of veins in the pterygomaxillary fossa and that thus air entered the circulation.

The air gets into the circulation in only one way. During the puncture a vein in the antral mucosa must be opened by the needle and the air directly forced into the vein, from which it proceeds by way of the facial and jugular veins into the right heart. It has been shown by Gürlitz that the veins of the antrum mucosa are numerous, and in 58 per cent of his well injected specimens there was a thick venous plexus over the nasal wall or orbital wall or over both walls. Whenever this plexus is present, therefore, it is present on exactly those walls of the sinus where it might easily be injured by the needle entering from either the inferior or the middle meatus.

Even in those cases of death in the literature in which no air was found in the circulation at autopsy, there might, nevertheless, have been present an air embolism in the coronary arteries or in the vessels of the brain and been overlooked at section, for, as Brauer says, the pathologists may easily overlook this factor, for few of them think of removing and opening these organs under water unless the clinician specifically requests it. In regard then to our death cases with negative section reports, those which have been previously reported as vagus deaths or cocain poisonings must also receive serious consideration as cases of air embolism.

I am in perfect accord with Boenninghaus, who does not deny that vagus excitations may arise from too strong an air pressure or too cold an irrigating fluid or what not, but does not believe it possible for this factor to function to such a degree as to produce actual death, and I feel that this must in each case be laid at the door of air embolism in the circulation, even though the section reports were negative.

The probability that a large number of my twenty-nine reported cases who escaped with their lives were also cases of air embolism rests upon the fact that during the inflation of the antrum with air certain nervous symptoms and manifestations appeared which Brauer found also appeared in his experimental work on the lung where he injected air into the pleural cavity. These nervous manifestations would arise during antrum inflation if the air pumped into the circulation were insufficient to completely fill the right heart and stop its action suddenly. The air would then be forced through the lungs and into the left heart and the greater circulation, and by passing into the vessels of the brain and the medulla would cause certain symptoms until it was absorbed.

In this connection Kelley says that the grave symptoms which arise from exporatory puncture of the antrum are strikingly similar to those which occasionally arise during the production of an artificial pneumothorax. The following symptoms have been observed after the latter procedure: pallor, dyspnea, cyanosis, unconsciousness, rapid pulse and respiration, diplopia, amblyopia, tonic and clonic contractions of groups of muscles, general convulsions, maniacal excitement, pareses and paralyses. Kelley quotes Lillingston as saying, "It is now generally admitted that the symptoms of pleural reflex are not clinically distinguishable from those of gas embolism; and even the necropsy may not be decisive, for a fatal dose of gas may have escaped from the blood vessels of the brain before these could be examined."

In 1912 Zesas had collected fifty-four cases of convulsions following operative measures on the pleura of which twenty-one proved fatal.

We see, therefore, that a close analogy exists between the symptomatology produced by forcing air into the antrum of Highmore and that produced by introducing air into the pleural cavity.

In general, therefore, it is only a question of how much air and how rapidly it is pumped into the circulation which decides whether our patient is going to die or recover.

Now, as to the explanation of my own case, I am in some ways quite at sea. The emphysema and edema of the orbital and periorbital tissues and lids can of course be explained on the basis that either the needle itself punctured the orbital wall or that there was present a bone dehiscence. This latter supposition does not seem reasonable to me in as much as this patient had been irrigated eight to ten times previously without mishap. The emphysema and edema experienced in this case has probably been experienced by almost every rhinologist and the usual course is for the swelling to subside after a number of days or weeks and leave no serious after-effects. This is exemplified by a large number of the cases from forty-eight to seventy-nine. In none of these cases was there any serious after-effects to life or to the organ of sight.

It was different, however, in my case. As soon as the eye could be observed after the accident it was blind. That this was of recent origin is evidenced by the fact that when the eye ground was first seen the disc was still of good color and only showed a progressive atropy in the following weeks. The eye condition was, therefore, definitely one of retrobulbar neuritis. To my mind this condition was due to the underlying sinus infection and not to the accident sustained at the time of irrigation. I cannot conceive of any possible condition which could be caused by the introduction of air into or around the orbit which would give us this result in the eye ground. Certainly not an embolism of the central artery of the retina, for this must be transient in character, as in the cases of Hirsch, Killian, Hajek, Streit, and Walliczek, and if by any possibility it could have caused a permanent thrombosis of the vessel it must have produced an eye ground picture such as we usually see in the thrombosis of the central artery of the retina. This typical eye ground picture was certainly absent in my case. I am still open to suggestions from any one as to just how this result occurred.

And now, with regard to the method of puncture employed, a formerly much mooted question, it does not seem to me that this has any application whatever, for we find that in the death series seven punctures were from the inferior meatus, three from the middle meatus and in five the route was not noted.

In the next group of twenty-two cases, with symptoms very similar to the death cases, there were twelve punctures from the inferior meatus, two from the middle meatus and in eight the meatus was not noted.

In the group of cases with transitory blindness there were three punctures from the middle meatus, two from the inferior meatus and in one the route was not noted.

In the last group of cases of local emphysema and infection there were ten punctures from the middle meatus, fourteen from the inferior meatus and in seven cases the route was not mentioned.

In the total of all the cases there were eighteen punctures from the middle meatus, thirty-eight from the inferior meatus, and in twenty-one cases the puncture was not noted.

The old bugbear of the possible danger of wounding the orbit by the middle meatus route has been very much overrated, for while there were in all twenty complications which might be considered ocular, orbital or periorbital in character, twelve of them were produced from the middle meatus, nine from the inferior meatus and in four the route was not noted; moreover, these cases were all minor accidents without a serious result in any one except my own, and I still question whether this was due to the operative procedure employed.

And finally, what are we going to do to make this valuable diagnostic and therapeutic measure safe? For it must be conceded by all that a procedure which has produced in a short number of years fifteen recorded deaths and many other serious complications, even though in thousands and thousands of other cases it has been used without serious consequences, is not perfectly safe. Are we going to discard the method entirely as Dutrow and Neugebauer advise, or are we going to try and obviate its dangerous features?

For avoiding the shock of the puncture itself in heart cases Kronenberg would weigh the indications with great care and when found absolutely necessary would place these patients in the reclining position for the puncture itself and then have them sit up for the irrigation.

When the middle meatus is filled with polyps these should first be removed. When the maxillary ostia are narrow or stenosed and the air meets with great resistance, Gording would have us make the opening in the inferior meatus sufficiently large for both air and water to freely pass through and when the wall of the inferior meatus is unduly thick and the needle might easily strip the mucosa in passing through he would have us choose the middle meatus route. Certainly in all cases the middle meatus should be thoroughly shrunk down before the puncture.

When he finds that blood returns through the canula Kayser, of Breslau, does not use air inflation at all but aspiration instead.

But why use air inflation at all? Because of his own case of air embolism of the central retinal artery and the other cases of air embolism in the literature, Caesar Hirsch has given up the inflation of air either before or after the irrigation and finds that his patients suffer no harm therefrom.

We usually use the air inflation before irrigation to determine if the point of our needle is in the sinus and after irrigation to dry out the sinus. I do not believe it is necessary for either of these purposes. If the needle has passed through the nasal and lateral walls of the sinus from the inferior meatus or through both the nasal and the orbital walls of the sinus from the middle meatus its shaft will not be movable. Should the point of the needle be imbedded in the mucosa then the slightest movement of it should cause pain, as would also the injection of the smallest amount of fluid. Neither is it necessary to dry out the sinus after irrigation. We certainly cannot hope to do more than mechanically cleanse the antrum by the act of irrigation, and it can do no possible harm to leave a small amount of salt solution, boric acid or any other mildly antiseptic solution in the antral cavity after it has been washed clean of the gross products of infection. Moreover, it very rapidly leaves the cavity by changing the position of the head or by blowing the nose or by absorption.

I have, therefore, myself abandoned the air inflation of recent years and have not seen any deleterious change in my results.

My conclusions are:

1. That we have in the puncture and irrigation of the maxillary sinus a very valuable diagnostic and at times also a valuable therapeutic measure.
2. That it makes very little difference whether you irrigate from the middle or the inferior meatus, but that irrigation from the middle meatus is probably easier on the patient.
3. That the procedure as formerly employed is not entirely without danger.
4. That while the anesthetic used and the question of vagus irritation cannot be entirely eliminated, the main danger consists in the air inflation and not in the act of puncture or the irrigation.
5. That we can make the procedure comparatively safe, at least eliminate the more serious complications, if we avoid the use of air either before or after irrigating.

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LIV.

SURGERY OF THE ETHMOID LABYRINTH.

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Many methods of operating upon the ethmoid cells have been devised. Some have been comparatively simple, only contemplating drainage of the anterior cells while other operations have been exceedingly complicated, contemplating complete removal of the middle turbinate and all accessible ethmoid cells. In most of the operations the cells have been attacked through the nose, while in some the extranasal method has been advocated. As a general proposition it may be stated that a physician's disposition to operate extranasally bears an inverse ratio to the development of his intranasal technic. In practically all the operations a part or all the middle turbinate has been sacrificed and the space formerly occupied by the turbinate and ethmoid cells is turned into the general nasal cavity.

It is not the purpose of the writer of this paper to present anything new or startling in the field of ethmoid surgery, but, first, to protest against the unnecessary sacrifice of the middle turbinate body and, second, to call attention to the method (not new) of draining and ventilating the ethmoids.

Several years ago the writer pointed out the tendency to maxillary antrum disease following the removal of the middle turbinate, and experience through the years has strengthened the belief that one of the functions of the middle turbinate is to protect the antrum, probably by leading discharge from the frontal sinus and anterior ethmoid cells away from the antrum and to cause the discharge to drop free into the nasal cavity. Another function of the middle turbinate is to direct part of the air current past the ostium maxillare in such a way that it will rarify and condense the air in the antrum with each inspiration and expiration. That the density of the air in the antrum varies with each expiration and inspiration can be readily demonstrated by the following cadaver experiment:

Remove the portion of the bone of the canine fossa, leaving intact the mucous membrane of the antrum. Then attach a Politzer's air bag to the posterior naris of the same side. Alternate pressure and relaxation upon the air bag will stimulate the action of the respiratory muscles, and the exposed mucous membrane in the canine fossa will be seen to retract and bulge in unison with the movement of the air bag.

That the middle turbinate and the ethmoid cells have a function in maintaining a proper lumen of the nostril is indicated by the fact that after removal the patient usually finds himself unable to prevent the discharge accumulating in the area once occupied by these structures. Continued crust formation is a common sequel. The discharge in this locality harbors and favors the development of microorganisms which frequently invade the adjacent cavities, the frontal, maxillary and sphenoid sinuses.

There is such a variation in the pathologic conditions in the middle turbinate and ethmoids that it is difficult to lay down explicit rules for the management of the different cases. The writer has no doubt that diseased turbinates may sometimes cause disease of the ethmoid cells and it was his practice formerly to remove the turbinate in the hope that the diseased ethmoids might recover. Later it has seemed that diseased ethmoids have been the frequent cause of diseased turbinates and the plan has been to operate upon the ethmoids in order that the middle turbinate might recover. There are sometimes abnormalities of the middle turbinates which require surgical intervention regardless of the condition of the ethmoid, but that is not the subject under discussion. It is frequently necessary to swing the turbinate away from the lateral wall in order to get at the ethmoids. Under thorough cocainization this is neither difficult nor painful. The instrument used in exenterating the ethmoid cells is the Knight's biting forceps. The instrument is opened about a third of an inch and while the shaft is pressed over against the septum the blades are introduced under the middle turbinate against the bulla ethmoidalis. Then with a gentle rocking motion the blades are pushed into the ethmoid labyrinth as deep as the cupped part of the blade (about three-quarters of an inch). The forceps is then closed and the tissue caught between the blades is removed. The forceps is inserted again, this time with the upper blade in the cavity made by the first bite and the lower blade below the floor of the ethmoid labyrinth. The instrument is pushed back almost to the anterior border of the sphenoid, the forceps is closed and the tissue caught between the blades is removed. Ventilation and drainage has now been provided for both the anterior and posterior cells, except possibly a few anterior cells about the nasofrontal duct.

The advantages of the operation are:

1. The ease with which the operation can be performed under local anesthesia.

2. The safety of the operation when the operator has a fair knowledge of the anatomy of the parts involved.

3. The thoroughness with which this comparatively simple operation exenterates the ethmoid labyrinth, as shown on the cadaver.

4. The after-results show no tendency to crust formation or other undesirable conditions. In examining some of the patients years after they were operated upon by this method no history of recurrence could be obtained and the only evidence that the patient had ever had ethmoid disease was the finding of an open space when the probe was inserted under the turbinate.

The dangers of operating are:

1. To the eye through the orbital wall, the lamina papyracea. With ordinary care, without unusual force, and by keeping the instrument perpendicular this accident should be easily avoided.

2. To the cranial cavity through the cribriform plate and the horizontal plate of the frontal bone. If the directions are followed as to depth of inserting the blades this accident cannot occur.

3. That the operation will not be thorough enough to effect a cure. Ventilation with the least possible interference with function is the object sought. Unless there is extensive necrosis the probabilities are that if this operation fails, the more extensive operations would also fail. Should this operation fail more extensive work can be undertaken at any time. It is well to remember, however, that it is always easier to take out more tissue if necessary, than it is to put back what has been removed.

LV.

THE PATHOLOGICOANATOMIC DIFFERENCE BETWEEN THE FETID AND THE NONFETID OZENA.

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Few laryngologic subjects have been discussed so industriously during centuries as the question of the ozena (as far back as the days of Galen we can trace the discussion of this question). Yet we find in the laryngologic textbooks that the authors still have not been able to determine which of the different theories of the ozena is correct.

What then do we mean by the ozena? That the ozena is a disease of the nose characterized by four main symptoms, i. e., fetor, atrophy of the mucous membrane and of the turbinate bones, presence of crusts, deformity of the nose, all authors agree. But to indicate the uncertainty which yet prevails among the laryngologists with reference to which of these symptoms are pathognomonic for the ozena, I want to quote from Krieg, the author of the monography of this subject in Heymann's "Handbuch der Laryngologie." He says: "Untersucht man viele Fälle von sogenanter ozaena, so findet man keine der Erscheinungen, die den Typus zusammenzusetsen scheinen, constant, sieht, dass sich jede gradeweise abschwächen kann bis zu vollständigem Fehlen, muss sogar zugeben, dass jedes einzelne Symptom—mit Ausnahme naturlich des Fötor—in das Gegentheil einschlagen kann, so dass jedes Symptom, welches man als eigenthümlich fassen möchte, unter der Hand in nichts zerrinnt, und man endlich in Verlegenheit gerath, wenn man sagen soll, was man unter Ozeana verstanden haben will. Diese Verwirkung herrschte in Alterthum, schleppte sich durch das Mittelalter hindurch und ist auch von der Neuzeit trotz Rhinologie und Bakteriologie noch nicht zu allgemein unbestrittener Klarheit aufgelost worden."

In order to get a solution of this much argued question, Prof. Alexander in Berlin, as is well known, in the year 1912 proposed a collaboration in this question among the laryngologists all over the world. This proposition was favorably accepted in laryngologic circles, and almost every laryngologic society appointed a committee for this purpose. The results of this collaboration have not yet proved very conclusive.

As I have been interested in this subject for many years, have made bacteriologic, pathologic and anatomic researches and have gathered clinical material, I published the results of my researches as soon as Alexander had sent out his proposition.

I understand very well that the history of the question of ozena is so well known to this society that it will be necessary only to mention the theories as the nature of the ozena. They are the following:

1. Ozena is a primary, genuine atrophy of the nasal mucous membrane due to an unknown cause.

This opinion was originally proposed by B. Fränkel, who also was the first one to emphasize that one must differentiate this form of ozena from syphilitic and tubercular processes in the nose with their deeper ulcerous destruction.

2. The theory of the diseased sinuses (O. Weber, Michel, Grünwald). Ozena, according to this theory, is caused by a chronic purulent inflammation in one or in several accessory sinuses of the nose.

3. The mechanical theory (Zaufal, Hopman, Saenger). Ozena is due to a congenital lack or to a rudimentary development of the turbinate bones.

4. The process is due to a specific microorganism (E. Fränkel, Loewenberg, Abel, Thost, Perez, etc.).

5. Ozena is caused by degenerative processes in the mucous membrane, particularly the transformation of the cylinder epithelium into pavement epithelium (Gottstein, H. Krause, Habermann, Siebenmann, Choleva and Cordes).

6. Ozena is a hereditary syphilitic process (Stoerk, Gerber).

When I began my researches on this subject, as far back as 1896, the bacillary theory was popular. The essays of E. Fränkel, Loewenberg, Thost and Abel had recently been published: especially was the essay of Abel alluringly written. My intention was then to try to find a vaccin for the ozena. I soon found that Abel's mucobacillus was nothing else than the pneumobacillus of Friedländer and was quite innocent as a cause of ozena. I succeeded in showing that one could find this encapsulated bacillus in the secretion in cases of other nasal diseases as well as in the secretion from the normal nose. Later on I extended my investigation to include also other microorganisms which Marano and Strazza, Belfanti and della Vedova claim to be the cause of ozena. My results were also here negative. Furthermore, I examined the crusts and the tissue slides from the mucous membrane and the turbinate skeleton. In the tissue slides I found the same pathologic changes which have been described by all other authors on this subject—i. e., interstitial inflammation, epithelial metaplasia and fatty degeneration. But by examining microscopically the crusts as well as the more fluid secretion I found a clew. I found a distinct difference between the crusts from the fetid and the nonfetid ozena. In the former I found, among pus cells, epithelial cells and other detritus masses, also a large number of drops of fat of unequal size. They occurred in such large numbers that their presence could not be explained only from the fatty degeneration. These drops of fat were not found in the secretion from the nonfetid ozena. How could we then explain the presence of these drops of fat? It was this observation that made me suspect that some changes in the bony tissue must take place in the fetid ozena, as it is well known that fat will be produced in no small degree where there is decay of bony tissue.

By that time a case of cured ozena happened to come under my observation. It was a man sixty years of age. During his childhood he had always been suffering from rhinitis. At 16 to 18 years of age this rhinitis changed character and fetid crusts were discharged from the nose. When he was 40 years old this secretion began to subside. When I examined him his nose was highly atrophic; both the concha inferior and the concha media were reduced to low ridges; the ethmoid cells were visible and lined with a thin, pale gray mucous membrane; the ostia of the sphenoid sinuses were visible and wide open. How is it possible that the atrophy could be the primary factor and the cause of the ozena if in a case like that, where there is atrophy in its highest degree, no ozena is present?

Furthermore, I have observed, as all other authors of this subject, that in all cases of ozena the sense of smell is lost. As the endings of the olfactory nerve are distributed in the region of the superior meatus the superior ethmoid cells and the upper part of the septum, I began to suspect that this region is the very seat of the disease.

Furthermore, when I tried to answer the question whether the secretion in the ozena is a secretion of the surface or a secretion of the sinuses I found that without any doubt a secretion of the surface exists besides the sinus secretion. It is only partly correct, as Grünwald says, that the secretion flows from the sinuses and dries to crusts in the nasal cavity. But if the crusts are carefully removed every day the secretion in the outer nasal cavity is gradually diminished. From this I came to the simple conclusion that the crusts irritate and keep up the secretion in the outer nasal cavity. This is not a trifling matter, as it first might have appeared, because it is this secretion, caused by the inflammation which the crusts create by the irritation, both mechanically and bacteriologically, that gives the ozena its characteristic clinical appearance and its name rhinitis atrophicans.

These observations led me to believe that we must look for some pathologic changes in the sinuses as the cause of the ozena, although they could not be found through the use of ordinary methods of examination.

At that time I happened to see a case of extreme ozena, which I have reported extensively in "Archiv. für Laryngologie und Rhinologie," Band 26, Hft. 1, Pg. 1: Group A, Case 1.—I opened the ethmoid cells, the sphenoid and frontal sinuses and drained them until they were permanently healed, whereupon the secretion and the formation of crusts and the fetor ceased. In this case the small operation conchotomia media fully convinced me that the theory of the diseased sinuses is correct: not an atrophy sui generis but a cario-necrotic process of the gravest kind was located in the walls of the sinuses. In the same paper I published all my cases of ozena and diseases similar to the ozena of the nose illustrating this question. There were all together 21 cases of fetid ozena, 10 cases of nonfetid ozena, 4 cases of partial atrophy in the nose and 4 cases of healed ozena. Of the 21 cases of the fetid ozena, the process was located in 13 cases in the ethmoid cells and the sphenoid sinuses on both sides. In one case the ethmoid cells and the sphenoid sinuses on one side were affected, in one case the ethmoid cells on one side, in two cases the sphenoid sinus on both sides, and in three cases the ethmoid cells, the sphenoid sinuses and the frontal sinuses on both sides.

The fact that with the healing of the sinuses, secretion, formation of crusts and fetor cease goes to prove without any doubt that the processes in the sinuses are the original causes of the ozena. Even if in some very severe cases complete healing cannot be established, this is naturally no evidence against the correctness of this statement. However, everything is not hereby explained as regards the nature of the ozena. To fully comprehend it we must also be able to explain the pathogenesis of the four main symptoms: the fetor, the changes of the outer form of the nose, the atrophy and the formation of the crusts.

The fetor is naturally explained as being due to a carionecrotic process in the wall of the sinuses. The correctness of this statement is furthermore proven by the fact that there is a distinct difference, pathologically as well as clinically, between the fetid and the nonfetid ozena. In the former there is, as mentioned, an ulcerous process with destruction of the mucous membrane and carionecrosis of the bony tissues. In the latter the mucous membrane of the sinuses is swollen, red and produces a mucopurulent secretion but otherwise intact.

In regard to the outside form of the nose, it is not a saddle nose. It has a broad base and a low bridge, but it is not sunken in, as in the case of ozena syphilitica. This is not only a gradual difference but a distinct difference in shape. The same observations that I have made concerning the cause of the peculiar shape of the ozena nose can be made with still less difficulty in case of tumors in the ethmoid cells, such as fibroma or fibrosarcoma. As the tumor grows, it pushes the middle turbinate bone against the septum and at the same time dislocates the lateral wall of the nose and pushes forward that triangular part of the face which is located between the base of the nose and the eye. By the dislocation forward of this triangular part, the ridge of the nose will appear to be lower. If the tumor is removed, the middle turbinate bone will still remain dislocated, but the lateral wall of the nose and the trianglar part above mentioned will sink back, the nose will be narrower, and the ridge of the nose will appear higher. In the same manner I have found that hypertrophic ethmoid cells in cases of younger individuals, where the tissues are soft and elastic, dislocates the parts mentioned. Now if the ethmoid cells are removed, one can notice the same change in the breadth of the base and the height of the ridge of the nose as in cases of tumor.

In reference to the atrophy the question arises: What is the cause of it and how does it develop? I have once before mentioned that B. Fränkel, who first described the ozena as a specific nasal disease, considered the atrophy to be the primary factor and the cause of the ozena. Zaufal, Hopman and Saenger, however, considered the atrophy, or, as they call it, "the atrophic condition," to be congenital. Later on B. Fränkel somewhat modified his opinion, particularly since Hajek published his cases. Insisting that the atrophy is primary, he says now that the same atrophic process may extend from the nasal cavity into the sinuses. Several of my cases have shown that the atrophy and the increased width of the nasal cavity are not always simultaneously present. One may find cases with fetor and wide meatus but without atrophy of the mucous membrane. Furthermore, one may find cases where certain parts of the membrane are atrophic, while other parts are hypertrophic in the same nasal cavity. This seems to me to show that a hypertrophy has preceded the atrophy. The presence of this hypertrophy I have also ascertained by examining microscopic slides.

These prove that an inflammatory process exists and that this is not always uniform, as I have found that this inflammation shows partly the picture of a purulent catarrh, partly the one of a round cell infiltration and partly the one of an indurative process with increase of the connective tissue. The last mentioned form I have found only in the more advanced cases. Consequently we find in ozena the same kinds of inflammations as we usually meet in the chronic and subchronic forms of rhinitis and sinusitis.

I have described how in younger individuals the outer form of nose is changed after the middle turbinate bones and the ethmoid cells have been removed. The effect of this change, the dislocation of the outer wall of the nose toward the median line, can be also seen in the nasal cavity, as one can see the lower turbinate bone move closer to the septum. By this dislocation the width of the nasal cavity will be made smaller. This observation shows that it is the hypertrophic ethmoid cells which by the pressure on adjoining tissue contribute to the increased width of the meatus. This increased width will later on be still more pronounced from the atrophy of the mucous membrane and of the skeleton of the turbinate bones.

The cause of the atrophy in the membrane is the crusts. Through the irritation these crusts exercise they keep up a chronic inflammation in the mucous membrane which finally causes the atrophy of the membrane. If the crusts are removed often and carefully one will see the secretion will be diminished. In case of partial atrophy one can see the atrophy only on those parts where the crusts have been located.

Besides these main symptoms we have another factor which has been claimed to be the cause of the ozena, namely, the degeneration of the mucous membrane, the change of its cylinder epithelium into pavement epithelium, and the horny degeneration of its outer layer. This symptom, it seems to me, is also secondary. It is a phenomenon which we see everywhere in the pathology that the mucous membrane exposed to a chronic purulent process will have its cylinder epithelium changed into pavement epithelium, as, for instance, the mucous membrane in the cavum tympani in cases of a chronic purulent process in the mastoid cells.

It has been said that one could not with certainty prove a carionecrotic process in the ethmoid cells, because the walls of the individual cells should be so easily broken and so easily penetrated by the probe that even in the case of normal cell walls one would get the scraping sensation which a carious bone gives and thereby get a deceptive result. In addition it has been claimed that one has not been able to prove microscopically the presence of carionecrosis. I decline to admit the correctness of this argumentation, because if we have ever seen a case like the one which I have described in group A, case 1, in the above mentioned report, we cannot possibly maintain that the normal and the carious ethmoid cells can be confounded. It is not, however, in the walls of the separate ethmoid cells that I have found this process, but in the walls of the evacuated ethmoid cavity and in the walls of the widely opened sphenoid sinus. The more extreme the cases are, the more extensive is the destruction and the more easily it is observed. But to remove all doubt I want to emphasize that in the freely opened sinus frontalis no one could very well assert that a mistake could be made between an ulcerous and a nonulcerous process. In the former case there is an exposed gray green miscolored bony surface and in the latter the sinus covered with a red, inflamed, thickened but otherwise intact mucous membrane.

From other sources it has been said: "I have also operated for sinusitis in cases of ozena, but there must be some other factor besides the sinusitis which causes the ozena. In the beginning there was a little improvement, but the conditions soon became what they were before." In these cases there must have been some affected sinus which has been left unoperated. Note this mentioned case 1 and how clear it is. An affected sinus which is not operated on leaves just as large and just as irritating crusts with just as bad fetor as before.

There is a great difference in the opinions of the physicians as to the seriousness of this disease, which is explained by the varying intensity of the disease. The milder cases cause little trouble, but the moderately severe cases already cause the patient great suffering, while in the extreme cases the suffering is so great that the disease takes all the pleasure out of life: yes, cases have been heard of where the bearer has tried to commit suicide. The severe headache, the general ill feeling and the loss of energy caused by the resorption of the septic and putrid substances never leave him. Furthermore, he may have some of the complicating diseases, such as the recurring otitis, the chronic pharyngolaryngitis and bronchitis and the rheumatic diseases which so often go with the ozena and add to the suffering. To this physical suffering the mental and social sufferings are added. In their working places the patients notice that people try to keep away from them, and they have a hard time getting a position. This causes timidity and hypochondria. The disease may cause the patient to sink to the lowest stratum of social life. See case 10.

In regard to the operations, there is not much to say about the ethmoid cells and the sphenoid sinus. The operative procedures of these sinuses in the ozena are the same as in ordinary cases. It is only of importance to open them as wide as possible. It is different in the cases of the frontal sinus. Here the ordinary methods of operation are not sufficient. In these cases we must remove the anterior wall of the sinus and all the carious bony tissue on the posterior wall. The cosmetic result of this procedure may not be the very best, but by the use of irritating applications the granulations may grow so as to fill the cavity. Fortunately the cases of ozena in which the frontal sinus is affected are comparatively rare.

I have several times in this paper mentioned the opinion of B. Fränkel that the atrophy is primary. It is unfortunate that this theory has been introduced, as it has in many respects prevented the solution of this question. Those who claim that the atrophy is primary must logically condemn every operative interference. Every opening of the sinuses and removing of the turbinate bones must, according to their opinion, be not only unjustified, but also distinctly contraindicated, as it would only augment the atrophy of the nasal cavity and facilitate the drying up and the decomposition of the secretion. The popularity which the treatment of ozena by injection of paraffin has gained shows to what absurdities the theory of B. Fränkel leads. By these injections of paraffin into the turbinates one endeavors to increase their volume and thereby decrease the tendency of the secretion to dry up. Even if in this way this object is gained, it is reached at the expense of the aggravation of the disease. The atrophy is nature's way to self-healing, if this self-healing even takes decades. The paraffin injections cause a barrier to the free flow of the secretion and thus the resorption of the septic material in increased.

It has also been said that, because experience has shown that this disease as time goes on heals by itself, one should not operate these cases. There is no doubt that some cases heal spontaneously, and personally I included four healed cases in my report. How large a percentage of the cases heal by themselves we do not know; presumably it is only the milder cases. In one of the cases reported by me the patient was not able to have her ozena treated. Eight years later I was informed by her sister that she died of meningitis. This does not indicate that the ozena is so harmless a disease as has been claimed.

It has been claimed that ozena is contagious (Strübing, Ziem. Perez) and that the contagiousness is evidenced by the fact that one often finds several cases in the same family. This observation I have also made, but I have always been able to trace its origin to some epidemic, as diphtheria or scarlet fever, as the common cause of these sinusitises.

In the beginning of this lecture I quoted Krieg as saying that although all authors agree upon the four main symptoms, it still is difficult to say what ozena is, because these symptoms are not always constant, as each one may by degrees diminish and even entirely disappear. This is a most telling remark. Considering the inconstancy and the gradual lessening of these symptoms, how easy is the origin of the secondary symptoms understood according to my explanation. One finds a perfect correspondence between the clinical symptoms and the pathologicoanatomic picture. In cases of intense fetor the carionecrotic process involves all the sinuses; in cases where the fetor is slight the carionecrotic process is limited to one sinus. In other cases an abundant secretion, forming crusts in both nasal cavities, entirely covering the walls, but without fetor, all sinuses are diseased, but there is no carionecrotic process. In another case the atrophy is of a high degree, but all other symptoms lack—a cured ozena. In another case no atrophy but hypertrophy, fetor, presence of crusts, and width of the meatus—an ozena of recent date.

So the picture can change kaleidoscopically—in one case we find only one-half of the symptoms, in another case only one-fourth, but they all are parts of the whole clinical picture of the ozena. Corresponding to the degree of clinical symptoms there is a sinusitis of smaller or greater extension, of younger or older date, of smaller or greater destructions.

It is more than 25 years since I began these researches. Every year that has passed, every case that I have studied during these years has only strengthened my opinions in regard to the nature of the ozena.

LVI.

ABSCESS OF THE LUNG AND THE METHOD OF PREVENTION IN NASOPHARYNGEAL SURGERY.

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There is no question that abscess of the lung has been too frequent a complication of operative intervention in the upper air tract. This is partly due to other important facts, namely, that in most of the operative procedures in the upper air tract we have to do with infected material, the escapage of which we are not always able to control, and whose escapage is facilitated into the area to be secondarily infected by a most perfectly arranged open tubular system.

The process of infection in the large majority of cases is hardly open to argument. Infection takes places in the larger percentage of cases through the direct inspiration of infective organisms, contained in fluids, pus, small particles of cheesy material, small masses of tissues or other debris. There is probably a small percentage of cases occuring after tonsillar operations wherein the infection takes place through the lymphatic or vascular system. There is no doubt that operative work done under the unconscious state, without a well conducted technic, is more productive of this occurrence than is the same operative procedure done under the conscious state.

We are, nevertheless, learning through reports of cases that operations done in the conscious state are not immune from pulmonary infection, as well as other forms of infection, which do not transpire under the unconscious state.

It would lead us into too long a discussion to give the reasons one may conscientiously have for preferring general to local anesthesia in faucial operations. Sufficient is it to state that the operation can be done safer, better and with less risk to the patient under general anesthesia. General anesthesia (ether) in the hands of a skilled anesthetist should be absolutely safe to life. Local anesthesia as applied to producing anesthesias in the fauces has already demonstrated that it is a dangerous agent to the life of the patient.

It is not my purpose to present the manifold reasons other than those generally outlined above why general anesthesia is preferable to local anesthesia in faucial operations; as the main object of this paper is to attempt to demonstrate how through carefully conducted technic under general anesthesia the occurrence of pulmonary abscess in faucial surgery can be eliminated.

The technic which I have adopted and which seems to have eliminated this type of surgical risk in faucial surgery is as follows:

1. A careful preoperative preparation of the patient.
2. A medical anesthetist, skilled in the use of the Beck or similar type of anesthetic and suction apparatus.
3. The placing of the patient in the moderate Trendelberg position during the operation. Head and shoulders well dependant.
4. The anesthetist skilled in following the knife and desector or other pressure about the fauces so as to gather up through suction pus, cheesy material or liquid exudate from the tonsils.
5. The most gentle manipulation in applying compression and swabbing.
6. The patient is returned to the bed resting on left side and should be placed in bed face downward until thoroughly reacted.

Through the careful carrying out of the above technic I have had a perfect relief from this unpleasant complication in faucial operations during the past four years, although I have performed more than my usual ratio of adult operations.

In the nasal and nasopharyngeal operation, which from their major character we are accustomed to resort to general rather than local anesthesia, it is not surprising that we occasionally have a lung abscess as a complication. Until the occurrence of this complication following some of these operations became a subject of comment, the operative protective technic was not universally employed. To safeguard our patients should be one of the cardinal principles in surgery. To do the operation well, skillfully and quickly is not sufficient. It is only through the employment of the greatest care that we can prevent leakage into the lower air tract.

In operating upon the antrum, frontal, sphenoid and postethmoid cells, filled as they are with infective material, it is imperative to block off all possible means of the escapage of pus, granulations or other debris into the lower air tract through properly placed postnasal tampons.

The nasal chambers should be well cleansed before the posttampons are removed. The posttampons should not be removed until the patient has reacted sufficiently to give evidence of self protection.

In the radical operation on the antrum through the canine fossa route, it is important to place a large tampon in the buccal cavity in order to prevent leakage from this area onward into the lower air tract. The patient should be protected by posture after returning from the operating room until he has completely reacted.

Pulmonary abscess is such a typical and thoroughly characteristic condition in its etiology and symptomology that one should not mistake it for any other disturbance. That such mistakes are, however, frequently occurring is the only excuse I can offer in closing my paper with the presentation of the symptomology of this disease. In most cases you have an operative procedure upon a more or less infective area in direct communication with the lower air tract. The invasion may occur immediately or be delayed three or four days after the operation has been done. If the invasion is apparently delayed, the patient does not do well in the interval between the operation and the first signs of invasion. The actual invasion is usually manifested by a chill or chilly sensation, followed by a rapid rise in the temperature, which immediately assumes a septic character. Pain in some area of the lung is early present. Cough paroxysmal in character is a very early symptom. Odor of the breath first noted by the patient, later by attendants, is quite a characteristic symptom. Profuse heavy pus expectoration, which may become rusty as the case progresses. Hemorrhage may occur. Profuse sweating at night. Temperature, pulse and respiration run in usual ratio. The earliest physical signs are those of an infiltrated area, frequently unrecognizable in the early stage, becoming more clearly defined on successive days. The radiograph gives an absolute picture which cannot be misinterpreted.

The most important element in treatment is early diagnosis. The early production of artificial pneumothorax is productive of the best results. If the abscess is discovered too late, illy situated for pneumothorax, or of such nature that rupture may take place under its application, then surgical methods must be employed in treating this unfortunate complication.

LVII.

HYPERPLASIA AND INFECTION IN POSTETHMOID-SPHENOID OCULAR COMPLICATIONS.*

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The ocular and orbital complications of paranasal sinus disease have always been of extreme interest to the ophthalmologist and to the rhinologist. Recent medical literature records many valuable observations, but there is yet much to be desired in the way of positive diagnosis, prognosis and treatment.

Anatomic studies impress one that the abnormalities presented by the cells of the accessory sinuses make clinical diagnoses increasingly difficult. The anatomic variations of the frontal and anterior ethmoid group are well known. They have their respective brain, orbital and ocular complications, but the difficulties of diagnosis and methods of procedure are not so great as when the cells of the superior nasal passage are involved. It is particularly to the latter and their ocular complications that this paper is devoted.

The ethmoid cells of the superior nasal passage are confined to no definite boundary lines. From their position normally in the wall of the superior meatus they may extend upwards and in forty-five per cent of all individuals there is a cell from this group in the wall of the first supreme nasal meatus.[1] These cells may occupy a large portion of the orbital wall extending even into the frontal bone. A pneumatization may take place centrally into the turbinates forming the socalled conchal cells, and laterally it may take place at the expense of the maxillary antra. Posteriorly, the sphenoethmoid cell may not only invade the body of the sphenoid bone, but may almost obliterate its sinus and extend even into the lesser wings of the sphenoid bone. There is thus established a remarkable variation in the anatomic relationship between the cells of the posterior ethmoid group and the optic nerve. The normal relationship is not extensively intimate because the general directions of the optic nerve and this group of cells diverge. An anomalous extension of this group of cells may, however, accomplish a nearly complete encircling of the nerve. Furthermore, the thickness of the bony wall separating the optic nerve and the posterior ethmoid cells varies from five millimeters to an actual dehiscence of osseous structure.

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*Candidate's Thesis, presented to the American Laryngological, Rhinological and Otological Society.

The fact that the fetal sphenoid sinus is entirely outside the body of the sphenoid bone and that its development is wholly a process of pnematization into the body of the bone, enables one to better understand why the adult sphenoid sinus presents so many anomalies. The extension of this pnematization is known to take place into the lesser wings, into the clinoid processes, into the pterygoid processes, into the basilar process of the occipital bone and into deviations of the nasal septum. The sinus itself is not a simple cavity, diverticula, incomplete and complete septa often being present. Schaeffer has recently called attention to the fact that the sinus may invade the great wing of the sphenoid beyond the point of the large foramina and that "in such cases the sinus and the temporal lobe are so intimately related topographically that one cannot ignore the sphenoid sinus as a possible factor in temporal lobe abscess."[2] The thickness of the bony walls may diminish to actual dehiscences of osseous tissue, thus bringing the mucous lining of the sinus into an extremely close relationship to the hypophysis cerebri, the optic nerve and the cavernous sinus with its contained structures. Sluder has directed particular attention to the intimate relationship between the sinus and the vidian and the maxillary nerves. These anomalies of such unusual frequency have been established by the work of Onidi,[3] Loeb,[4] Schaeffer,[1] Sluder,[5] and others. We are without clinical means of gaining definite preoperative information as to many abnormalities which may be present in the cells of the superior nasal passage. Even operative measures fail to give one complete and definite data as to anomalies that may occur.

Radiography reveals more or less definitely anomalies of the sinuses in the body of the sphenoid bone. Some have attempted to outline and interpret by this means anatomic variations of the posterior ethmoid cells but as a general diagnostic aid the X-ray is often only of relative value. The presence frequently of so many anatomic abnormalities makes it difficult or impossible at times to establish a diagnosis by clinical examination. The fact that any long continued inflammation of the cells of the superior nasal passage rarely confines itself to one of the two subdivisions, sphenoid and ethmoid, may be a matter of some consolation to the operator, who from his clinical examination might feel that he was needlessly sacrificing cells of the posterior ethmoid group in order to investigate, with greater ease and thoroughness, a pathologic condition known to be present in the sphenoid sinus.

The etiologic factors to be considered in the orbital and ocular complications are: firstly, the spreading of infection or inflammation, suppurative or nonsuppurative, by direct extension; secondly, the transference of infection by the venous and lymphatic channels; thirdly, the absorption of toxins from infected cells; and fourthly, the results of mechanical pressure. Fortunate as it is for the patient, it is rare that the opportunity occurs to study these cases at autopsy and thereby substantiate or disprove the various theories advanced.

Orbital cellulitis, a direct spreading of inflammation, is nearly always due to accessory sinus disease. R. E. Wright[6] states in a paper on this subject that in children this condition is more often a primary acute affair, while in adults it is more frequently an exacerbation of a chronic disease. Naturally such a condition arising from cells of the superior nasal passages would give symptoms developing from an involvement of the tissues deeper in the orbit. Skillern quotes Gerber[7] as stating that under favorable conditions, purulent inflammation of mucous membrane may cause the breaking down and ulceration of bone. Thus we may have an orbital abscess, meningitis or brain abscess with actual dehiscence of bony structures. Extension by contiguity may also exist where the bony walls have apparently suffered no inflammatory change, yet under the microscope are shown to be hemorrhagic and infiltrated with the infective organisms.

The intimate intermingling of the blood circulation between the ethmoid and sphenoid sinuses and the orbit and the interior of the cranium is well known. Not only through the anterior and posterior ethmoid foramina is this interchange established, but the lateral and inferior walls of the orbit are frequently pierced by diploic veins which arise in the walls of the sinuses, and all eventually reach the cavernous sinus through the ophthalmic vein. From the side and roof of the sphenoid sinus similar veins penetrate the bony wall to reach the coronary and cavernous sinuses. Bordley[8] has emphasized this vascular intimacy regarding involvements of the optic nerve—firstly, in that the papillomacular bundle of the intracanicular portion of the nerve lies immediately in contact with the vein of Vossius, and secondly, that the vein of Vossius, which drains the intracanicular portion of the nerve and empties into the cavernous sinus, receives numerous branches from the periosteum of the posterior portion of the orbit and also from the periosteum lining the sphenoid and posterior ethmoid sinuses. Based on known histologic relationships abundant opportunities are offered for the occurrence of ocular and optic nerve complications through the venous channels in the form of phlebitides and the means for a direct transference of toxins.

Lastly, there must be considered the eye and particularly the optic nerve complications due to pressure. The fact that constant pressure of soft tissues will displace bone justifies one in assuming that pressure due to the presence of a mucocele or pyocele may be the all important factor with such complications. True mucoceles are rather rare. Skillern states that up to 1916 only six cases of mucocele of the sphenoid sinus had been reported and that these were all associated with a posterior ethmoiditis.

The theory that hyperplasia of the mucous membrane of the accessory sinuses with or without accompanying hyperplasia of bone, particularly in the apparent absence of suppuration causes retrobulbar neuritis, edema and papillitis and even choked disc, has brought out marked differences of opinions between rhinologists, neurologists and brain surgeons. The work of Sluder and Wright has been an incentive to the making of observations by others; but, because the clinical diagnosis of such cases is so fraught with the personal equations of the commentators one is left quite undecided as to the actual status of hyperplastic tissue. Furthermore, such observations have so often failed to distinguish between the hyperplasias per se and the hyperplasias with suppuration that the conclusions reached have really not clarified the problem. Bordley[8] states that about thirty per cent of the cases of sphenoid and ethmoid disease show optic nerve involvement. An opinion at variance with this is held by Beck[9] in reporting ninety-one patients suffering from accessory sinus disease, including nineteen cases of atrophic rhinitis and ozena, who failed to show any unusual deviation from the normal in the way of scotomata and defects of the visual fields. In no case of empyema of the sinuses was there substantiated a connection between disease of the optic nerve and the accessory sinuses, the conclusion being that the importance of the topographic relationship between the posterior ethmoidal and the sphenoid sinuses and the optic nerve has, in its clinical aspect, been overestimated. Viewing the subject from the ocular symptoms rhinologists have more or less generally accepted the fact that an enlargement of the blind spot was practically pathognomonic of postethmoid sphenoid disease. The converse does not hold true. Bordley[8] found an enlargement of the blind spot in but thirty-one per cent of a series of one hundred two cases suffering from postethmoidal sphenoidal disease, though this percentage was materially increased in those suffering from acute fulminating inflammations. Fish[10] has reported nasal accessory sinus disease to be present in thirty-six successive cases of optic neuritis. Cushing[11] states his belief that a hyperplasia of mucous membrane is incapable of producing a retrobulbar neuritis with swelling of the disc equal to two or three diopters and accompanies his remarks with a rather severe criticism concerning the number of nasal operations being performed to relieve such conditions.

The hyperplasias attending chronic postethmoid sphenoid disease and their status relative to the cause and effect of ocular complications has been the basis for the greatest differences of opinion at the present time. It is acknowledged now that polyps and polypoid hypertrophies may be due to external causes and that many of the underlying bone changes are the result and not the cause of the pathology in soft tissues. So it may be conceded that the hyperplasias of the lining membranes of nose and accessory sinuses are the result of external irritation and that the accompanying bone changes are secondary to and the result of the same. The type of external irritation is, however, often overlooked, its mechanical cause being taken for granted, and yet we may have hyperplasias in which the irritating element is of bacteriologic origin. The pressure of hyperplastic tissue without macroscopic evidence of infection should not establish the presumption that the eye complication is entirely due to pressure. The cases of spontaneous recovery and relief following operation rather militate against the contention that the complication is entirely a matter of mechanical pressure.

A review of some of the case reports in the current literature reveals that the eye complications in the reported observations have dealt largely with pathology due to or accompanied by purulent conditions. Sluder's observations have stimulated interest in the hyperplastic conditions, and his statement that the optic nerve complications from pressure in the hyperplasias usually develop slowly should constantly be kept in mind. In the following condensed report of twenty-two of his cases, notations are made as to the eye complication, the presence of hyperplasias and secretions, and the results obtained.

1. Dilated pupil, hyperplasia present, suppuration, relieved by treatment.
2. Dilated pupil, hyperplasia present, dry, treatment nil, relief by operation.
3. Paralysis of accommodation, hyperplasia, pus, recovery after operation.
4. Scotoma scintillans, intense inflammation, no hyperplasia or pus, operative relief.
5. Photophobia, hyperplasia, intense inflammation present, recovery by treatment.
6. Trochlear paralysis, acute sphenoiditis, recovery by treatment.
7. Iritis, sphenoid suppuration, operative recovery.
8. Iritis, postethmoid sphenoiditis, recovery by treatment.
9. Uveitis, postethmoid sphenoiditis, with secretion, recovery under treatment.

10. Punctate keratitis, postethmoid sphenoiditis, with secretion, recovery by operation.

11. Acute blindness, hyperplasia, pus, recovery by operation.

12. Failing vision, hyperplasia present, dry, thickened bone, results nil.

13. Transitory amblyopia, suppurative sinusitis, operative improvement.

14. Transitory amblyopia, hyperplasia with secretion, operative recovery.

15. Choroiditis, postethmoid sphenoiditis, pus, operative improvement.

16. Choroiditis, postethmoid sphenoiditis, pus, operative recovery.

17. Retrobulbar neuritis, sinusitis present, relief by operation.

18. Optic neuritis, hyperplasia, pus, bone showing rarifying process, improvement.

19. Optic neuroretinitis, hyperplasia, dry, results failure.

20. Optic neuroretinitis, sphenoiditis, secretion present, recovery.

There are but four cases, which from clinical examination, did not reveal the presence of infection. Three of these had to do with the optic nerve and in only one of these three was there slight improvement. The other two remained unimproved. The co-existence of suppuration or secretion with hyperplasia in the cases obtaining relief by the establishment of drainage is striking.

Vail[12] reports a case of postethmoid abscess with exophthalmos and third nerve palsy with recovery following operation.

J. A. Cavanaugh [13] makes a rather significant report as to bacteriologic cultures taken from the sphenoid sinus in fifty cases with obscure eye symptoms. None of these cases were of the acute suppurative type but were insidious in nature. Where the cultures were negative there was no improvement in the eye symptoms following the opening of the sphenoidal sinus.

Bordley[8] reports five cases. First, acute blindness, from suppurative sphenoiditis with operative recovery. Second, loss of vision and divergent squint with suppurative postethmoid sphenoiditis, no relief from operation. Autopsy revealed a necrosis of the orbital wall with extradural abscess. Third, choked disc and central scotoma accompanied by an intense neuralgia with negative findings by intranasal examination and operation. Autopsy revealed a double sphenoid sinus with pus present only in a posterior compartment. Comment: the anatomic anomaly here was largely responsible for the failure to diagnose this condition at operation. Possibly the taking of radiographs with the probe in the sphenoid sinus might have helped to establish the anomaly in this case before operation. Fourth, double choked disc and ocular palsy (external rectus) with no pus discovered by clinical examination. Pus was found at the time of operation which was followed by the recovery of vision in one eye. Fifth, central scotoma and enlargement of the blind spot; pus being revealed by suction. Relief was afforded by this method of treatment for two years. Recurrence then necessitated an operation, which was followed by recovery.

R. C. Hamill[14] reports a case of recurring diplopia due to a suppurative sinusitis with relief by operation.

B. M. Dickinson[15] reports a case of vitreous opacities and failing vision with a nonsuppurative hyperplasia of the nasal mucosa. There was present a deflected septum and the X-ray showed a darkened antrum with pain localized over the same. Septum resection, exenteration of the ethmoid cells and opening the antrum gave relief.

G. B. Jobson[16] reports four cases of impaired vision accompanied by suppurative postethmoid sphenoiditis, and, following operation, three recovered, while the fourth, which had been blind for one year, showed an atrophic nerve head and did not improve.

C. G. Crane[17] reports two cases of optic neuritis, the first showing a marked suppurative postethmoid sphenoiditis, while the second revealed only at the time of operation the presence of polyps and a mucoid secretion. Both recovered following the operations.

E. M. Sykes[18] reports three cases, one of blurring of vision, one of recurring spells of blindness, and one of unconsciousness following headaches. The author's diagnosis was hyperplastic sphenoiditis or postethmoid sphenoiditis, but all were accompanied by pus. All recovered following operation.

T. R. Boggs and M. C. Winternitz[19] report an unusual case of suppurative hypophysitis as a complication of purulent sphenoiditis which was without eye complications. The clinical examination of the nose was negative, the various sinus openings appearing normal and only by postmortem was the real condition discovered. The case here simply illustrates what dire complications may result with an infection present, and that this infection may remain undiscovered even with the ostia appearing normal.

J. Watson Williams[20] observed three cases, one of inability to use the eyes and two of mental dullness with headache, in all of which a mucopurulent discharge was demonstrated coming from the postethmoid sphenoidal region. Recovery followed in all three cases.

E. C. Ellet[21] gives an account of two cases of optic neuritis. The first patient having an associated suppurative post-ethmoid sphenoiditis underwent an operation and recovered. Examination of the second likewise revealed pus. The operation was delayed, and there being no clinical evidence indicating infection of the cells of the upper nasal passage, the operation when performed was limited to the frontal and anterior ethmoidal cells. This operation was not followed by improvement in vision, though the swelling of the nerve head was reduced.

Bordley[8] in reporting a series of eleven cases of optic nerve disturbance in which postethmoid sphenoid operations were performed with return of vision to normal, notes that in four cases no pus was found at operation, but adds that there was definite evidence of inflammation. Relative to the presence or absence of pus there was no difference in the degree of improvement in these cases. No notations were made on the bacteriologic findings.

L. E. White[22] reports a series of twenty-five cases and believes that pressure on the optic nerve from hyperplasia is sufficient to cause the optic nerve complication, much more often than is generally acknowledged. The duration of the nerve complication does not seem to be considered in determining his diagnoses. In fourteen of his twenty-five cases he attributed the nerve disturbance to hyperplasia. In one of these, with an X-ray finding of a hazy sphenoid, there was spontaneous recovery and the middle turbinate was removed after vision became normal. In three cases the cause was attributed to an extension of inflammation and in eight cases to toxemia due to the presence of pus. In six of these eight cases the pus was not discovered prior to operation. If the diagnosis of hyperplasia pressure has to be made largely by exclusion then the most careful means must be taken in our clinical examinations before attempting to positively establish the same.

This rather incomplete resume of case reports from recent literature substantiates the countless reports of former observers who established the fact that pus or infection in the postethmoid sphenoidal cells may be a cause of optic nerve complications. We are still more or less in doubt as to the exact role played by hyperplasia of tissue in this region. As Jonathan Wright[5] states, this question is concerned more with a specificity of locality rather than a specificity of pathologic activity. After discussing the bone changes following chronic postethmoid sphenoiditis, he says, "that it probably would be difficult to find an adult individual in temperate or cold climates who does not present an example of this bone change (referring to the osteoblastic and osteoclastic activities) within his nasal chambers, which we have a right to call pathologic."

If it is a pressure from bone change that is responsible for the optic nerve complication, then it is inconceivable how an operation which can not reach the structures involved is able to bring about recovery. Such a causative factor is limited to optic nerve changes of slow progress and in which the operative results will be nil or at least will not bring about a restoration of function. If the nerve changes are due to pressure from soft tissue it should then be remembered that we are attributing to a chronic condition, practically ever present, the ability to rapidly cause extensive nerve disturbances. Here also there is no direct operative attack on the tissue about the nerve and, considering the rapid recovery so often observed, it seems that there must be some other element present besides pressure. The spontaneous recoveries reported tend to strengthen the idea that pressure alone is not wholly responsible. It is generally conceded that the involvement of the optic nerve takes place in its intracanicular portion. It is rather difficult to attribute only to pressure from without, a selective action, which will in one case cause only an enlargement of the blind spot and in another, central or paracentral scotomata. It seems more probable that if pressure alone is to be held responsible it will be confined to those cases developing complete blindness after a certain length of time. This, of course, is not intended to include cases in which there is a markedly acute inflammatory condition superimposed upon a chronic hyperplasia.

The limitation of hyperplasia of soft tissue to the above class of cases does not absolve from culpability hyperplastic conditions of the postethmoid sphenoidal region. First, we have a nerve extremely vulnerable to toxins and infections. Toxins and infections may exist for a long time and yet exert little influence on tissues within close proximity providing the avenue of drainage is not occluded, but if such avenues are closed, they can slowly or with extreme rapidity affect proximal and more distant structures. As stated previously we have here a close intermingling of venous channels. Pressure from within as well as from without is a possibility. Following infection the cellular infiltration may manifest itself by an inflammatory reaction and swelling, and this in a locality where the nerve involved is enclosed in a nonyielding bony ring. Again, toxins are capable of a selective action, which is amply demonstrated in other parts of the body, and it seems reasonable to presume that they may exhibit this same ability when involving the optic nerve. Infection and the production of toxins are not necessarily accompanied by pus. The normal configuration of the parts and the numerous anatomic anomalies of the same often make the demonstration of pus impossible either by clinical examination or operation. The rapid benefits derived at times by the establishment of drainage from a focus of infection are generally accepted. It seems reasonable to conclude from the remarkable clinical results, often quickly obtained, that the role of the hyperplastic mucosa here is more often one of obstruction to areas which harbor infection. While clinically this seems plausible, yet bacteriologic studies have not as yet fully established this presumption.

The most severe critics of the position taken by the rhinologist do not deny that postethmoid sphenoiditis is accompanied sometimes by optic nerve complications. Nor does the rhinologist deny that the same eye symptoms may be caused by intracranial pathology, and he feels that each case demands full neurologic findings. The latter may remain vague for a long time, and time is a most important element from the standpoint of the patient's recovery, particularly if the cause of the trouble lies in the postethmoid sphenoid region. The nasal operation, a drainage operation, is not one of great operative risk, nor is it apt to be particularly harmful to the individual so far as his future comfort is concerned. Moreover, infection, even of a suppurative type, is frequently revealed only at the time of operation. So the fact that patients who may have undergone a postethmoid sphenoid operation may later become charges of the neurologist or brain surgeon does not necessarily justify a criticism of the rhinologist. In the absence of positive neurologic findings in optic nerve involvements, clinical results certainly justify the rhinologist in establishing drainage in the postethmoid sphenoid region, not only when there is evidence of infection, but also when the only evidence is a possible lack of proper drainage due to hyperplasia of the mucous membrane.

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LVIII.

SOME PHASES OF ESOPHAGEAL STENOSIS.*

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Through the development of methods of direct examination of the lower respiratory tract, the laryngologist has broadened his field by turning his technical skill to the study of esophageal conditions with the aid of the esophagoscope. We claim justification for this theft from another province in the progress we have made in the diagnosis and treatment of esophageal lesions.

The clinical report that I am to make might be termed a study in diagnosis, for I seek here only to bring out some diagnostic features, and to show how important esophagoscopy is in determining the nature of a stenosis in this region.

Some six years ago I presented a thesis for admission to this society, in which was discussed simple inflammatory stenosis of the esophagus. At that time I could find very little in the literature of this subject other than some pioneer contributions by Guisez, in France, and Jackson, in this country. Their experience agreed with mine in that there might slowly develop a chronic stenosis of the esophagus from a spasm due to local irritation, which might result in a thickening of the esophageal walls and stenosis that could prove dangerous to life and yet be nonmalignant. Since publication of this essay I have had an opportunity to observe several additional cases, the most interesting one of which came under my observation the year prior to publication of the thesis mentioned. The diagnosis here is perhaps debatable, but the case is submitted under this classification.

May 1, 1914, Mr. M., aged 58, came to me with a history of fullness in the throat and difficulty in swallowing solid food, dating back some seven years. He had not been losing weight, having always been thin. Sometimes food was regurgitated several hours after eating. There was no pain at all.

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Esophagoscopic examination showed an annular constriction beginning at the lower portion of the upper third of the esophagus and extending downward several inches. This constriction yielded to dilation with olive shaped metal bougies, and after this was repeated several times Mr. M.'s condition seemed to be normal, as there was no further difficulty in swallowing. He was cautioned as to the possibility of return of the condition, and advised to consult me should he again experience difficulty in swallowing.

I did not again see this man, he having drifted entirely from my professional horizon, until September 1, 1921, when I was asked to see him, he being under the care of a local physician at the Baptist Memorial Hospital, the expediency of doing a gastrostomy being then under consideration and my advice requested.

I found Mr. M. bedridden, emaciated and very weak. He told me that since having been treated by me he had experienced complete relief for quite a while afterward, but difficulty in deglutition again developing, he began to receive treatment at various hands. Two physicians had diagnosed his condition as malignant, and he had received two courses of X-ray treatments. There had been no improvement whatsoever from these treatments, and for some time he had been able to take only fluids and not much of these. He was losing weight rapidly, but there was no pain. Radiographs showed an annular stricture, apparently five or six inches in length, beginning about midway of the esophagus and extending downward toward the cardiac end. There was considerable thickening of the involved portion of the esophagus, apparently fibrous in character. The radiographer at the hospital, without having previously heard my opinion, expressed himself as believing that the trouble was of a simple inflammatory nature. On esophagoscopy I found a characteristic constriction of the esophagus, as described, there being a perfectly smooth surface the entire length of the stricture, as determined by the use of a small sized Bruenings' esophagoscope. Wassermann was negative.

Immediately I began the practice of bouginage with Jackson's linen bougies, introduced through the esophagoscope. After the first treatment of this kind Mr. M. was enabled to swallow much more freely than formerly, and in ten days was able to leave the hospital, walking without difficulty, had gained in weight and strength, and returned to his occupation of farming in Arkansas. About once in two weeks he returned for bouginage and had no difficulty in eating even such solids as beefsteak.

Spending most of his time in the malarious delta land of Arkansas, Mr. M. fell victim to a severe attack of malaria, a recrudescence of a chronic condition, accompanied by a pleurisy, and about the middle of February died. A few days previous to that he had been in my office, and that morning had eaten an enormous breakfast, without difficulty, and had gained five pounds in weight in two months.

Allow me now briefly to emphasize some of the important features of this case. A diagnosis of malignancy, which had been made on two occasions, was perfectly natural without esophagoscopy, and without bearing in mind the duration of the condition. Lacking an autopsy, it is true that we cannot absolutely exclude carcinoma, but I have never seen an esophageal carcinoma lasting fourteen years, have never seen one entirely free from pain, and have never had a case that I could not dilate and bring up to almost normal deglutition, as I did in this case. The diagnosis of carcinoma was arrived at owing to the man's age, and the fact that he had a stricture of the esophagus. Here the value of esophagoscopy was evidenced. The esophagus showed no ulceration, no budding from the walls and no irregularity. Through the esophagoscope, and without anesthesia, dilation of the stricture was comparatively easy, and the use of such methods of precision obviated a wholly unnecessary gastrostomy.

A case of more than passing interest is that of B. C. L., male, aged 58, who came to my service at the Memphis General Hospital, February 25, 1922, with the following history:

Two months ago he began experiencing difficulty in swallowing. At that time he weighed 150 pounds. The difficulty in deglutition rapidly grew more pronounced, and he lost appetite and was greatly weakened. Everything he ate was immediately regurgitated, even fluids being largely rejected. He suffered with chronic constipation and at the time he came to the hospital had not had a movement in five days. Diagnosis made in the receiving ward of the hospital was partial pyloric obstruction. Wassermann was four plus. Radiography showed a stricture of the lower third of the esophagus, annular in shape, with no apparent thickening of the walls. There was also comparatively little dilatation of the esophagus above the stricture. The opinion of the radiographer was that the stricture was benign, and spasmodic in origin, with a cause elsewhere located.

Esophagoscopy was done by me on the morning of February 28th. The man was very emaciated and so weak as almost to be moribund. A large sized Bruenings' esophogoscope was passed down to the constriction with great ease, as there were no teeth to obstruct. A small amount of 10 per cent cocain was used to anesthetize the gums and hypopharynx. The picture clearly seen through the esophagoscope was that of a typical, uncomplicated esophagismus. There was no ulceration and apparently no thickening of the walls. It was easy to pass a size 30 Jackson linen bougie through the stricture into the stomach, and the man was so greatly emaciated that the end of the bougie could be felt through the wall of the stomach. The opinion expressed by me was that the esophageal stricture was a spasm, with a deeper and more serious cause. I could not conceive of such great emaciation and weakness from an ordinary, uncomplicated cardiospasm of but two months' duration.

Four days later, the man steadily growing weaker, he again was esophagoscoped by me, but nothing different seen from the previous examination. I very carefully swabbed the walls of the esophagus, but obtained not even one drop of blood. The next day at 11 o'clock he died. Just before he died he vomited a quantity of coffee ground material.

Autopsy by the pathologist of the University of Tennessee showed postmortem esophageal findings identical with those of the esophagoscopy, but the pyloric end of the stomach was quite another story. Here was found partial obstruction, a large amount of thickening around the pylorus and large ulcers of the pylorus and of the gastric mucosa in the immediate vicinity. The condition was regarded as syphilitic, and the tissues studied in the laboratory bore out this finding. There was no tumor formation. The thickening around the pylorus was an inflammatory fibrosis, and miliary gummata were found scattered throughout. The pathologist was convinced that the esophageal stricture was functional and secondary to the syphilitic process from below.

In the absence of a carefully worked out pathologic study of this case it would be perfectly natural, in the presence of extreme emaciation, epigastric pain, an esophageal stricture and a cancerous age, to make a diagnosis of cancer, and at the regular Monday evening conference of the staff of the Memphis General Hospital, before the report of the pathologist had been submitted, the trend of opinion expressed was to this effect, but both the gross and microscopic pathology negatived this. The esophagoscope had its value here in determining the character of the esophageal stenosis.

Now turn to another picture: Mrs. S., aged 45, was seen May 22, 1919. About four years previously she began having symptoms like "heartburn," accompanied by difficulty in swallowing. This difficulty in swallowing gradually grew worse until it became constant. At this time she could swallow only fluids and no acids. She had lost a great deal of weight, weighing 132½ pounds, whereas her normal weight was 175. A diagnosis of cancer of the esophagus had been made, but she was fluoroscoped ten months previous to the time that I saw her and cardiospasm diagnosed by the radiographer. The obstruction was at the lower end of the esophagus, where she at times had intense pain. Under fluoroscopy the barium milk could be seen to slowly trickle through the constricted cardiac end of the esophagus.

With a local anesthetic Mrs. S. was esophagoscoped and a stricture was seen, annular in shape, at the cardiac end of the esophagus, this apparently being two or three inches in length. No attempt was made at this time to go through the stricture.

Subsequently, at intervals of two or three weeks, the esophagus was dilated with Sippey's metal olives, some improvement following, and food being swallowed more satisfactorily, but in May of the following year, after the lapse of several months without her having been seen by me, Mrs. S. returned with the statement that she was again experiencing much difficulty in swallowing, was losing weight rapidly and had great pain at the epigastrium.

Under ether a Jackson esophagoscope was passed clear through the stricture and into the stomach. The next day Mrs. S. began to eat full meals and continued to do so, and in a few weeks' time had gained twenty pounds. Nearly a year later she reported that she was doing well. Mrs. S. then removed to Oklahoma, and a few months ago some difficulty in deglutition again returning, she consulted a surgeon in her locality, and I get the vague report that he said her trouble was reflex, from some uterine disease, did a vaginal hysterectomy, and Mrs. S. has had no difficulty in swallowing since. Veritably a neurosis!

A haphazard diagnosis of carcinoma perhaps was justified in this case, even more than in the one first related, for the symptoms were more indicative of carcinoma. Without the findings of the esophagoscope and the relief by its use this woman probably would have died of starvation from inanition. Spasm of the esophagus is rather common, but cases of spasm with the symptoms evidenced in this instance are not usual.

Another type of esophageal stenosis is exemplified in this case: A man, aged 26, came to me with a history of increasing difficulty in swallowing, over a period of two years. He dated the beginning of his trouble from a tonsillectomy, but this of course was to be ascribed to the human weakness to blame something or somebody for their ills. Pieces of salad and dry food were swallowed with difficulty. There was no regurgitation of food and so far no loss of weight. No pain whatsoever.

Esophagoscopy revealed nothing abnormal. The tube passed with constriction from only the regular anatomic narrowings. But it was different when barium milk was swallowed and its passage observed with the fluoroscope. The barium was seen to fill a small pocket, about the size of a hazelnut, in the esophageal wall. This was a pulsion diverticulum, and when filled exerted pressure, which caused the esophageal stenosis.

These four cases of varying nature have been selected from a not inconsiderable number of cases that I have esophagoscoped to determine the cause of stenosis in such cases. They also serve to emphasize how absolutely essential a carefully made esophagoscopy is in determining the true character of many of these strictures. In esophagoscopy we have annexed a new field of work, but to its development by laryngologists is due most of the progress made in the diagnosis and treatment of esophageal strictures, no matter how caused.

Before closing this clinical report I should like to urge that all cases of esophageal stenosis, no matter of what character, be investigated through the esophagoscope, and with this accurate means of study and of treatment in our hands there is very little excuse for the blind methods still so commonly practiced.

Bank of Commerce Building.

LIX.

INTERESTING BRAIN ABSCESS OF RATHER LONG DURATION.

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I wish to report the following case of chronic brain abscess which recovered after operation with complete restoration of all functions. I am not taking any undue credit for my success, as I understand fully that the abscesses which usually recover are those in which the stalk can be located, as I was fortunately able to do in my case. Nor do I claim that this complication in my patient was unusual in that it presented many symptoms not generally seen in this type of case. There were, however, one major and two or three minor features, which, if not exceptionable, are at least interesting.

The history is as follows:

Mrs. A. J., age 48, referred by Dr. M. S. Cotton of Goldsmith, Ind., April 21, 1920. Family history negative. Personal history: Lobar pneumonia 1905; complete recovery. Otherwise, with the exception of the left otitis media and the present illness, has never been seriously ill and always mentally and physically able to take care of household duties. Had an acute suppurative otitis media left in 1900 which lasted two or three weeks, and apparently became well. Since that time has had frequent attacks of acute suppurative otitis media left which appeared with little or no pain and healed without any treatment in a few days. Two years ago, following one of these attacks, the discharge did not cease and has continued more or less profusely up to the present time. Until the present illness has not suffered with headache.

Symptoms presented started seven weeks ago and were extremely violent, consisting of severe headache on left side of head and back of the left eye, nausea and projectile vomiting. There was extreme weakness of the extremities, and patient has been confined to her bed constantly. Patient becomes dizzy when in a standing position, with the sensation of falling backwards even when supported. The condition of the patient has depended absolutely upon the freedom with which the pus drains from the ear. It was discovered each time the drainage was very free there was a complete remission of all symptoms, but when there was almost a complete cessation of the discharge there was a return of symptoms.

Examination: Patient was a frail and emaciated woman, inclined to profuse explanations when answering questions, and was not able to enunciate her words clearly. She was rational, however, and able to give definite information when answering questions. Complained of a very severe headache on left side of head, double vision, weakness of lower limbs and inability to move the tongue towards the left. Pupils small, due to large doses of morphin, equal in size and responded slightly to light. She swallowed freely and remembered names of people and of places well. She was not able to retain food and vomited frequently and violently.

Patient stated after her recovery that several years previous to the operation she had not been able to smell, but following the operation she recovered this sense.

No examination of the labyrinths was made. There was no spontaneous nystagmus present. Hearing, A. D. 20/20, and A. S. 15/20. The scalp was slightly tender, but there was no tenderness anywhere over left mastoid process or edema of the superficial tissues. The left external auditory canal was partially filled with foul smelling pus coming from a perforation in the membrana flaccida posteriorly. There was very little drainage of pus from this ear. The right ear appeared normal. Reflexes were present and not exaggerated. No disturbance of skin sensation. There was no paralysis of any of the extremities. Visual fields were not taken at this time nor were the fundi examined. There was a partial paralysis of the left sixth nerve and a partial left Bell's palsy. I found very little increase in temperature; pulse rapid and weak; respiration normal. Because of the patient's poverty and lack of free laboratory facilities, no laboratory tests were made and the help of a neurologist was not secured.

Operation: A radical mastoidectomy was done the afternoon of April 21st. There was a complete sclerosis of the mastoid process, and the bone was found to be of ivory like-density down to a small antrum which was full of pus and granulations. The sinus, which was located forward, was accidentally uncovered and found to be normal. The roof of the antrum was necrotic, and its removal exposed the dura to the extent of the diameter of a dime. There was slight bulging. No pulsation. Because of the extremely poor condition of patient at this time, no further operative measures were taken and she was returned to bed. It is needless for me to say that I felt certain an abscess was present and would have made a search for it if the patient's condition had been better.

Following the operation there was a decided improvement of all the symptoms with the exception of the headache, which, while not quite so severe as before, still required sedatives for its relief. The patient was able to take and retain some nourishment but continued very weak. The temperature at no time following the operation became higher than 101.4; following the drainage of the abscess it dropped to 97; later returned to normal. Up to May 6th, the pulse did not become higher than 108 or lower than 72. Respirations remained around 24 until just before the abscess was drained, when they became weak and rapid. Spinal puncture was not done any time during course of illness.

At no time during this postoperative period was the patient in a condition for further operative work. Aside from dressing the wound, from which there was a moderate amount of discharge, relief of the patient's pain and such stimulative measures as thought necessary, nothing else was done.

On May 6th, about 3 o'clock in the afternoon, patient became semicomatose. Patient completely collapsed at 5 o'clock and was found pulseless. She rallied without stimulation and pulse taken again at 6 o'clock was found to be 64.

When I saw the patient the next morning she was almost completely comatose but responded slightly to pain. Nurse stated that she had not been able to feed her for the last twelve or fourteen hours on account of inability to swallow. Patient fell out of bed during the night. Involuntary evacuation of bowels and bladder. Pulse at this time was 47, good quality; respiration 20; temperature 97; slight right Kernig; beginning optic neuritis left eye.

Patient's condition continued to grow worse as the day advanced, and her death was expected almost any time. Pulse dropped as low as 40; temperature became subnormal and respirations very weak and rapid, of the Cheyne-Stokes character. There was some slight improvement that evening, and the pulse at midnight had increased from around 50 to 88. The next morning the patient was much better in every way. Seemed to be a trifle more conscious; temperature normal, and pulse and respiration, while weak, were nearer normal. The dressings were found to be saturated with a large amount of foul pus. Nothing was done at this time aside from dressing the wound, as it was decided that any operative interference might prove fatal.

The next morning, while exploring with a probe, I discovered a fistula in the roof of the aditus ad antrum, located just anterior to the exposed brain tissue. This I dilated very gently with a tonsil hemostat and drained from an abscess cavity about an ounce of pus. A drainage tube was inserted and the wound lightly dressed. This was done without anesthesia of any kind. During the dilatation of the fistula patient collapsed, but prompt stimulation resuscitated her. That evening the patient, while still somewhat cloudy mentally, aroused from her stupor and recognized those around her. Pulse and respiration were of much better quality and patient was able to swallow and was given some nourishment.

On May 10th, there was a decided improvement. I discovered that day that the patient had a typical paraphasia. The left pupil, which had been dilated for the past two days, was down to normal. Swallowed easily. There was a profuse discharge from the drainage tube.

May 17th, I removed the drainage tube, as there had been no discharge for the last four or five days, but a slight return of all her symptoms after twenty-four hours forced me to reinsert it. From this time on there was a continuous improvement, and the drainage tube was not removed, except for daily sterilization, permanently until June 17th. Following this the wound healed with absolutely no recurrence of any symptoms. Paraphasia gradually disappeared.

During the latter part of her illness I discovered she had an amnesia. She stated later on that after returning home she could only remember two proper names for several days (her two youngest children). She learned and remembered names again very rapidly when she could see them written but could not remember spoken words. Undoubtedly her auditory center for words was impaired, the visual center having escaped.

Following the exacerbation of her symptoms, when I removed the drainage tube May 17th, I decided to examine the interior of the abscess cavity with a Holmes nasopharyngoscope. I did this in order to discover when I would be warranted in removing the drainage tube. Just before the drainage tube was permanently removed I repeated the procedure. It was easily done, and I believe without any harm to the patient. It was not possible to tell whether there was a destruction of the convolutions that bounded the fistula, as when the drainage tube was removed the walls of the fistula would collapse. It was my opinion, however, that the fistula was between convolutions. It was between two and three cm. in length and pointed upward and inward at an agle of 60 degrees and very slightly forward.

The cavity was circular and was somewhere around 6 cm. in diameter. Its interior was bounded by convolutions of normal color, without any apparent break in their continuity. In the sulci I discovered what appeared to be cholesteatomatous material and argyrol which I had been injecting into the cavity at each dressing. At neither time did I discover any pus in the cavity nor any collapse of the cavity walls.

1. The major interesting feature of this case was the spontaneous evacuation of pus through the fistula after the mastoid operation. The violent symptoms presented after the beginning of her illness followed when the discharge became scanty. Twice between this time and when I first saw her the symptoms were relieved when the drainage was good and were increased when the drainage was poor. This case, I believe, supports Balance's contention that it would be wise in chronic cases to search for the stalk.

2. Another interesting feature was the absence of meningeal symptoms, with the exception of the right Kernig. This was evidently due to the sealing of the meningeal spaces around the fistula. This case is an argument in favor of producing

this condition before the abscess is opened if it does not already exist. In a review of the literature accessible to me covering the past ten or twelve years I noticed that where the abscess was quickly discovered and easily drained, with a minimum of trauma, the patient usually recovered. That where many incisions had been made through the brain cortex with a great deal of resulting trauma, even though the abscess cavity or cavities were successfully drained, the patient usually died.

3. Previous to the drainage of the abscess I did not discover any aphasia symptoms, unless it was a very slight interference of the motor tract. Following this procedure and while the abscess was draining freely a typical paraphasia put in its appearance.

4. The abscess must have been close to two years old. The history, together with the noncollapse of the cavity for several weeks following its drainage, to my mind supports this conclusion. That a macroscopic inspection of the cavity did not reveal any apparent destruction of tissue does not, I believe, militate against it.

5. Undoubtedly the abscess resulted from a chronic suppuration in the posterior part of the attic. This focus of infection certainly existed after her otitis media and must have been responsible for her repeated attacks of this disease. If cases of this type would submit to operation sooner brain complications certainly would become fewer.

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LX.

A SEPTIC TYPE OF TEMPERATURE NOT REFERABLE TO THE EAR IN CASES OF ACUTE SUPPURATIVE OTITIS MEDIA.*

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If a patient with acute suppurative otitis media has a septic type of temperature the natural tendency for the otolaryngologist is to assign the cause of the fever to extension of the infection from the ear and mastoid to the sigmoid and lateral sinuses. It has been well established in such cases that in taking time to make a differential diagnosis the physician does not endanger the patient. Such a course may reveal involvement of other structures to account for the clinical picture and thus danger to the patient and embarrassment to the physician may be avoided. It is not assumed that some absorption may not take place from the ear, but it may be assumed, from the clinical course of the case, that the ear has not been the important factor.

In the five cases reported herein it is believed that four disease conditions acted as causal factors in the septic type of temperature. It will be noticed that all the patients were young children, in whom the clinical course for any disease process may not be expected to run true to type, thus making diagnostic possibilities difficult.

REPORT OF CASES.

Case 1 (A358689). Acute Suppurative Otitis Media, Pyelitis.—Marguerite A., aged nine months, was examined in the section on pediatrics of the clinic May, 1921, because she had not been well for about a week. The mother had noticed that the child had a high fever every afternoon and was very restless at night. She had lost 5.5 pounds. The stools had been green. Cough had not been noticed.

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*Read before the annual meeting of the American Laryngological, Rhinological and Otological Society, Washington, D. C., May, 1922.

Examination revealed a temperature of 102.5 and a suppurative otitis media. The leukocytes numbered 41,000. Microscopic examination of the urine showed alkalin reaction, many pus cells and, on culture, bacillus coli. All symptoms disappeared under appropriate treatment for the pyelitis. The condition of the ear resolved after paracentesis and the usual treatment.

Case 2 (A335868). Suppurative Otitis Media, Central Pneumonia.—Alfred M., aged thirteen months, was first seen in the ear, nose and throat section of the clinic in September, 1920, when a suppurative otitis media was found in both ears. In November, 1920, the child was reexamined and trouble again found in both ears. He was kept under observation in the outpatient service. The records show that the ears would remain dry for one week, but without closure of the perforation, and the next week a discharge might be present. Early in January, 1921, the ears were almost normal.

January 14, 1921, the patient was brought to the Clinic because he had not slept well during the night and had often put his hand to his right ear.

Examination revealed the tonsils to be injected, the nose and left ear normal, and a dry perforation in the right ear, the membrane being milky white in color. The child's temperature was not taken, but two days later it rose rapidly to 104, and a mucopurulent discharge appeared in the right ear. The child was placed in the hospital for observation. During the night the temperature dropped to 99. The possibility of infection of the lateral or sigmoid sinus was thought of, but the presence of a slight cough made an investigation of the chest advisable. The examination was negative, January 20, the leukocyte count was 24,000; the examination of the urine was negative; culture of the blood was negative. Roentgenograms of the chest showed a definite shadow. The child was treated for the pneumonia. The condition of the ear did not change materially. After twenty-two days the child was dismissed from observation. (Fig. 1.)

Case 3 (A348892). Acute Suppurative Otitis Media, Abdominal Postoperative Condition.—Marcella K., aged four years, was brought to the Clinic February, 1921, because of a biliary fistula, following an abdominal operation elsewhere.

The child's temperature was 99. The biliary fistula was draining. The cervical glands on both sides were enlarged, the tonsils were slightly reddened, the nose and ears were normal, the chest was normal. Examination of the urine was negative. The leukocytes numbered 6,000; the differential was negative. A culture of the discharge from the biliary fistula showed streptococcus and bacillus coli. In a few days the temperature rose to 103. the patient complained of abdominal distress, and developed acute bronchitis and suppurative otitis media on both sides. The leukocytes then numbered 8,900; the differential was negative, and culture of the blood was negative. The increased temperature with chilly sensations indicated sepsis. Examination of the blood for plasmodium malariæ was negative; typhoid fever was ruled out. The condition of the ear appeared satisfactory. The chest remained negative. Further investigation elicited the fact that the child had had attacks similar to the present illness with chilly sensations and fever. No real causal factor was ascertained except the fistula in the abdomen. The child was dismissed from observation against the physician's advice after twenty-six days. Subsequent correspondence with the mother reevaled that a chiropractor was treating the child and that the biliary fistula had closed after two weeks. She had increased temperature occasionally (Fig. 2). A definite diagnosis was not made in this case.

Case 4 (A349322). Suppurative Otitis Media, Gastrointestinal Disturbance.—Theodore B., aged twenty-two months, was brought to the Clinic in February, 1921, because he had not talked, because he was extremely restless at night and had a "head cold." He had had whooping cough at the age of ten months.

Examination revealed definite evidence of rickets and an upper respiratory infection, the temperature was 100. Laboratory examinations were negative.

March 7th, the child was referred to the ear, nose and throat section because of earache. Suppurative otitis media of both ears was found and paracentesis tympani performed. The child was not hospitalized, because of the objections of the parents. There was an afternoon rise in temperature. All laboratory and physical examinations were negative. The parents were having difficulty in feeding the child and his bowels did not act normally. Castor oil was given and the temperature became normal and remained so.

Case 5 (A315116). Suppurative subacute otitis media, pyelocystitis.—Alice P., aged six years, was brought to the Clinic in May, 1920, because of a discharging left ear and fever in the afternoons. The trouble had started four weeks before with measles. There had been no chills. While the temperature was down the child appeared to feel well, although the general malaise was evident.

Infected tonsils and adenoids with enlargement of the cervical glands were found. The right ear appeared normal. The left canal was filled with purulent discharge. The membrane was full; the landmarks were not discernible. The discharge was pulsating through a posterior inferior perforation. There appeared to be no drooping of the superior canal wall, and tenderness of the mastoid was not elicited.

Many moist râles were heard throughout the chest. The temperature was 99. The leukocytes numbered 27,000; the differential count was negative. The urine contained many pus cells. The patient was sent into the hospital for observation, and treatment was directed to the condition of the ear and the pyelocystitis. As the urine cleared, the child's temperature and general appearance improved. The condition of the ear cleared up under local measures.

GENERAL SUMMARY.

It is apparent that in this group of children four clinical causes other than the disease of the ear were accountable for the clinical course. At least, treatment directed to the conditions appeared to clear up the symptoms. The deeply situated central bronchopneumonia in Case 2 was the most difficult to diagnose. Operation in this case no doubt would have resulted fatally. The hardly discernible cough was the symptom which led to the search of the chest for the cause of the symptoms.

The differential diagnosis in Case 3 was established on the fact that the chills and fever appeared shortly before the onset of the trouble in the ear and because of the infectious process in the abdomen, although previously there had been no chills and fever from this source.

Pyelitis, or cystitis, particularly in little girls, may be overlooked unless a microscopic examination of the urine is made. Helmholz (personal communication) says that it occurs with upper respiratory infections and that a diagnosis cannot be made unless a catheterized specimen of urine has been examined. He believes that excoriation and redness of the vulva and acid reaction of the urine are not the cause of the rise of temperature but the result of the temperature. It has been our experience, however, that alkalizing the urine with potassium citrate has caused these symptoms and signs to disappear (Case 5).

Gastrointestinal disturbances in young children often are the cause of a rise in temperature, particularly if a change in diet has been necessary because of a change in location. With other causes not discernible a brisk cathartic, such as castor oil, is sometimes effective. This procedure is not looked on with favor by many pediatrists.

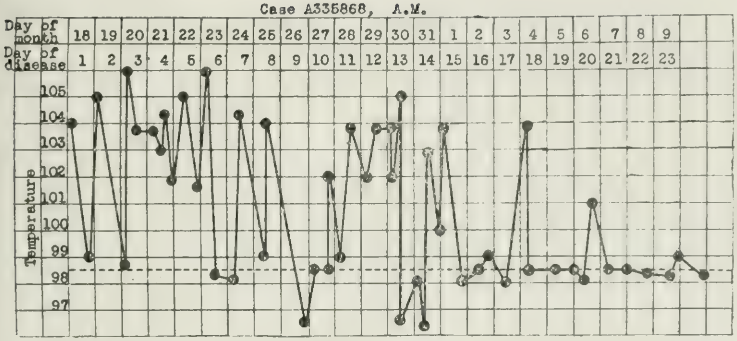
Case A335868, A.M.
Day of month
18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9
Day of disease
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
Temperature
105
104
103
102
101
100
99
98
97

Fig. 1.

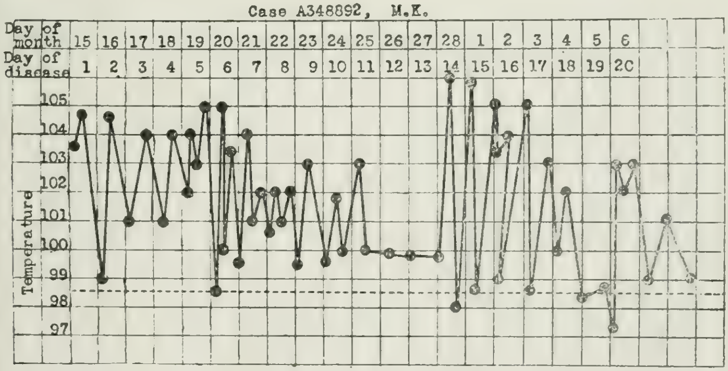
Case A348892, M.K.
Day of month
15 16 17 18 19 20 21 22 23 24 25 26 27 28 1 2 3 4 5 6
Day of disease
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
Temperature
105
104
103
102
101
100
99
98
97

Fig. 2.

LXI.

EXTRADURAL ABSCESS COMPLICATING FRONTAL SINUSITIS—REPORT OF A CASE.*

By Ross Hall Skillern, M. D.,

Philadelphia.

This case occurred in a young man of eighteen years, who on September 20th, went in a swimming pool, dived frequently, and on the following day had considerable bilateral discharge from the nose; otherwise well. This continued for several days. He then went swimming again with more diving. The following morning he was worse, headache frontal, considerable discharge from nose, yellowish, thick, with fever.

Following day (third day after last swim, October 5th) the upper right eyelid began to swell; other symptoms progressively worse. The same evening a probe was passed into right frontal sinus through nose, which was followed by some relief to headache, but swelling on lid and over right forehead became even more marked. The following morning suction was used and a considerable amount of pus was brought down, followed by gradual relief from pain, although tenderness and swelling remained. Next day, pain still absent, suction was again applied, with success as far as obtaining pus was concerned; tenderness and swelling became more pronounced. Suction every two hours during the night.

The eye had become entirely closed on account of the swelling and that over forehead greatly augmented, but still no headache; tenderness and fever remained well marked. Taken to hospital; ointment applied to eye and forehead. Suction continued once daily.

Condition about the same for four days, when forehead was incised, which was followed by the evacuation of two ounces of pus. Swelling and tenderness was relieved in the forehead but not in the eye. Suction treatment was continued. One week later upper eyelid incised, considerable quantity of pus evacuated. The swelling and tenderness were not relieved. Both fistulas continued active up to the time the patient was brought to Philadelphia (about ten days). In the meanwhile tenderness over the eye and forehead almost disappeared, but swelling remained over the eye; forehead considerably diminished.

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*Read before American Laryngological Association, 1922.

Examination disclosed no apparent swelling over the supraorbital region. There was some proptosis of the upper lid, right side, with discharging fistula about in the center. A second fistula directly over the middle of the eyebrow, also in an active state of discharge, was noted. Considerable tenderness on pressure but no apparent loss in the continuity of the anterior sinus wall was apparent.

X-ray examination: Right frontal sinus opaque, outer wall appears to be broken up. Right ethmoid region solidly opaque. (See skiagraph.)

Considering these findings, an immediate operation was of course decided upon, which was performed the following day, November 2nd.

Operation: After the usual curved incision through the eyebrow and down over the ascending process of the superior maxilla, on elevating the periosteum over the frontal sinus a loose sequester of bone was noted and removed, thus exposing the sinus cavity, which was filled, not only with pus, but detritus composed of granulation tissue and some particles of bone. The entire anterior wall was then removed. Bleeding was very free and required considerable sponging and pressure in order to make it possible to proceed with the operation. It was then observed that the posterior sinus wall, instead of being firm and unyielding, appeared to give way under pressure and pus would constantly ooze up after being sponged away.

Further examination disclosed a large defect in this wall, ovoid in shape, about the size of a five cent piece, at the bottom of which lay the exposed dura. The dura itself, in the center, was covered with a false membrane and surrounded with granulation tissue, and from the upper portion purulent secretion was constantly escaping. On inserting a director upward, between the dura and the bone, a sudden gush of pus covered the field, which proved to be a large extradural abscess with two ramifications, one almost in the median line, the other to the temporal side, each extending superiorly about 1½ to 2 inches. The posterior bony wall (which was badly necrosed) was then removed from below upward, over the body of the abscess, and finally over the prolongations, until the limits were accessible to the probe. All necrotic bone was removed and the edges curetted, the inferior sinus wall resected, and as no evidence was present that the subdural structures were involved the operation was completed as usual.

Iodoform gauze strips (one inch seamed) were inserted into the abscess cavity between the bone and dura, the whole cavity packed with iodoform gauze and the wound allowed to remain open, with the exception of the inferior portion along the side of the nose, which was closed with four sutures. The operation lasted a little over one hour, the patient leaving the table in good condition, pulse 102, temperature 98. The following day he appeared considerably shocked, was apathetic, and lay very quiet; pulse 120 to 130, temperature 100 3/5 to 102 degrees.

The next day (second after operation) the wound was dressed, the iodoform gauze packing partially renewed and fresh gauze inserted. General condition much improved, although very weak.

Bacteriologic report of the culture from brain pus showed staphylococcus pyogenes aureus in pure culture. The dressings were changed every second day, healthy granulations began appearing in depths of the wound.

On November 18th, under nitrous oxid oxygen anesthesia, the outer half of the wound was closed by four stitches of fine silkworm gut, and the cavity, which had greatly decreased in size and partially filled with healthy granulation tissue, again packed with iodoform gauze.

The second closure was made on November 23rd, and two stitches from the first closure removed, as they had become infected.

On November 28th, it was noted that several of the stitches from the second closure had cut out, so three new ones were inserted and the patient discharged from the hospital, just four weeks from his admittance.

The formation of an extradural abscess is due to an especially virulent infection quickly ulcerating through the mucosa and attacking the bone, and is further shown by the rapid appearance of fistulas. The posterior wall, composed entirely of compact bone, offers more resistance to the infecting organism (streptococcus or pneumococcus), but ultimately succumbs, thus laying bare the dura. This structure shows great resistance to infection and endeavors to throw a plastic wall around the purulent process by forming adhesions between the dura and bone. It depends largely upon the type of the infecting organism whether this will be successful or whether the infection will continue to spread and ultimately pierce the dura and infect the meninges and brain.

If the primary organism (streptococcus or pneumococcus) survive, the probabilities are that the infective process will become widespread, resulting in death. Whereas, should the primary infecting germs become choked out by a secondary infection (staphylococcus pyogenes aureus), the pathologic activities will tend to localize themselves within certain limits or at least show a tendency to spread very slowly, thus permitting well directed surgical intervention, coupled with rational after-treatment, to bring about resolution of the parts with complete recovery of the patient.

Frequency.—Gerber[1] in 1909 was able to collect but 28 cases of extradural abscess which were unaccompanied by any other form of cranial infection. However, three of these were complicated by subdural and frontal lobe abscess, and the records of eleven others difficult of access on account of having been personal communications or published as theses.

Some repetition also seems to have occurred, as four cases are credited to Laurens. In addition to these, I can add eight more, 2, 3, 4, 5, 6, 7. It would seem curious that frontal lobe abscess occurs much more frequently, if one can judge from the literature (Boenninghaus[8] reports 82 cases), as it is nearly always combined either with extradural abscess or some form of meningitis, but this can be explained if we consider the extradural abscess, not as the terminal stadium of the infection but rather as an intermediate process which is not arrested by nature or a surgical intervention will penetrate the dura and progress to the formation of a subdural and eventually frontal lobe abscess or general meningitis, as the case may be.

Extra dural abscess occurs much more frequently as a complication of acute frontal sinusitis than of chronic. This can probably be explained by the fact that if a patient has suffered for a long time with an uncomplicated case of frontal sinusitis his system builds up a certain general resistance to the infecting organism which, while not sufficient in itself to entirely throw off the infection, nevertheless is able to hold it in check as far as further spreading is concerned. An acute exacerbation can upset this equilibrium with all its liabilities of contiguous inflammation.

The symptoms are more often insidious, so that signs may fail entirely and the lesion incidentally found during the sinus operation which hitherto had been entirely unsuspected. This circumstance has been noted and commented upon by several authors,[5] [9] and obtained in my own case. Perhaps one of the most significant symptoms is the sudden appearance of swelling in the upper eyelid during the course of a frontal sinusitis, as it is rarely seen with uncomplicated cases and is practically pathognomonic for a rupture in one or more of the walls.

The headache is of little diagnostic value, as it may be conspicuous or entirely absent. If, however, it continues in severity out of all proportion to the apparent pathologic condition, then suddenly ceases, it is very significant that a rupture has taken place through one of the walls, with sudden cessation of the secretory pressure. The condition of the pulse also cannot be taken as a criterion. Pressure symptoms apparently do not occur.

The sudden onset of a chill during the course of a frontal sinusitis followed by high fever (104 to 105 degrees) is an unfavorable omen and can be taken for an extension of the infection, either by progress upwards between the dura and bone, by perforation of the dura, or the development of a septic thrombophlebitis (rare).[10] If the rise of temperature is accompanied by vertigo, bilious vomiting, rigidity of the neck and somnolence, the onset of meningitis or an encephalitis (brain abscess) has made its appearance.[8] Convulsions of a severe type have been known to be caused by simple irritation of the frontal lobe.[11]

Pathology.—The infection in intracranial complications may spread in the following manner:

1. Through continuity.
2. Through the bone (canaliculi).
3. Through blood and lymph passages.
4. Through dehiscences, causing the following pathologic sequence:
    1. Pachymeningitis externa (circumscribed).
    2. Extradural abscess (circumscribed).
    3. Pachymeningitis interna.
    4. Intradural or subdural abscess.
    5. Leptomeningitis (diffuse).
    6. Encephalitis (brain abscess, frontal lobe).
    7. Thrombophlebitis (sinus thrombosis).

These can in nowise be considered separate and distinct pathologic processes, but rather a continuation of the infection, provided it has not been arrested at one of the stages by natural (resistance of the organism) or artificial (surgical intervention) means.

As far as the pathologic process for the intracranial infection is concerned, it would appear as though spreading by continuity, together with the perforating veins, play the most important role. In this way, the perforation of the bone is caused by the pressure of the secretion on the mucosa, thus furthering ulceration and necrosis of the periosteum. The vena perforantes carry the infection through to the meningeal side, setting up a periostitis at that point. The bone, thus being deprived of its nutrition on both sides, a localized solution of continuity occurs, resulting in a necrosis, followed by sequester formation. The osteomyelitis, with softening, may extend around the venous passages for some distance.

Paunz[12] believes that an extradural abscess forms when the underlying bone becomes slowly infected, thus permitting adhesions between the dura and bone. My case does not bear this assertion out, and I believe the virulence of the organism exercises great influence upon the subsequent course of the infection. If the original infecting germ (streptococcus or pneumococcus) continue to thrive in pure culture not becoming attenuated, the case will probably go on to general meningitis and death, whereas, if a secondary organism (staphylococcus) finds entrance and chokes out the original causative factor, the chances are much better that the infection will tend to localize itself, thus becoming amenable to treatment and saving the life of the patient. This fortunately is not a rare occurrence, as it has been shown that the staphylococcus is present in complicated cases of frontal sinusitis twice as often as the streptococcus.[13]

I cannot entirely agree with Dreyfuss[14] when he states that if a perforation of the posterior wall is found, the chances are one to two that a brain abscess is present, but I can endorse his statement that extradural abscess is more frequently found in widespread necrosis (infection of the diploë) than in simple perforation of the posterior wall.

Treatment.—Drainage is the absolutely essential factor in order to bring one of these cases to a successful conclusion. This can best be accomplished by iodoform gauze and a wide open wound. As these extradural abscesses are usually unsuspected and found by chance during the sinus operation, we must always be ready to deal with one should that eventuality arise. Fistula formation in the upper lid or the supraorbital space should invariably be looked upon with the greatest suspicion. Necrosis of the posterior wall is practically always accompanied by a circumscribed leptomeningitis or, if the purulent stage has been reached, by an extradural abscess. When this is found the posterior wall must be removed until the limits of the abscess are freely accessible to the iodoform gauze packing. Most authorities insist upon the removal of the bone until it is flush with the limits of the abscess cavity. This is not necessary and certainly not indicated, particularly after the superior limits of the sinus cavity have been reached, for after the posterior wall has been resected to its upper limits further resection will only bring the scalp into juxtaposition with the dura, which will shut off the dural space more completely than the hard, unyielding wall of the frontal bone, not to mention the permanent defect to the bony covering of the brain which follows.

When indications point to the presence of dural involvement, the posterior wall should be opened where it appears suspicious and the dura freely exposed. If it is discolored, nonpulsating or covered with granulations or false membrane, pus should be sought for by introducing a probe between it and the bone in various directions. When the abscess has been found (which usually occurs immediately if present), the limits are carefully sought for by the probe, as one must be careful not to break down any adhesions which may be forming between the inner table and dura to wall off and localize the collection of pus from the general meningeal cavity. The posterior wall is then carefully resected with the heavy bone forceps, usually in an upward direction, until the limits of the abscess cavity can be reached. Seamed iodoform gauze strips are packed loosely between the bone and dura, and after the inferior wall of the sinus has been removed and a large counter opening made into the nose, the entire cavity is packed with iodoform gauze and only that part of the incision alongside of the nose closed. The outer dressings are changed in 24 hours, as they become saturated with blood, which dries and becomes very stiff and uncomfortable. The internal dressing is changed on the second day, but the gauze strips between the bone and dura are left undisturbed. Dressings are subsequently changed every two days until the sinus cavity begins to become filled with healthy granulation tissue, when the gauze strips can be removed (about one week after the operation). Partial closure of the wound can now be made with silkworm gut sutures but a loose packing still kept in the sinus. After a week or so this packing can be discontinued and the entire wound closed, provided good drainage has been established with the nose.

If the symptoms do not subside or an augmentation of the same takes place within forty-eight hours after the operation, the wound should be opened to ascertain if by chance a damming back of the secretion has occurred. If not, the dura should be immediately incised and the frontal lobe explored for the possibility of a brain abscess. Freudenthal[15] reports a case that four days after a radical frontal, on account of continuation and exacerbation of symptoms, the wound was again opened and the dura incised, which was followed by the escape of 100 grams of pus. Complete recovery followed in six weeks.

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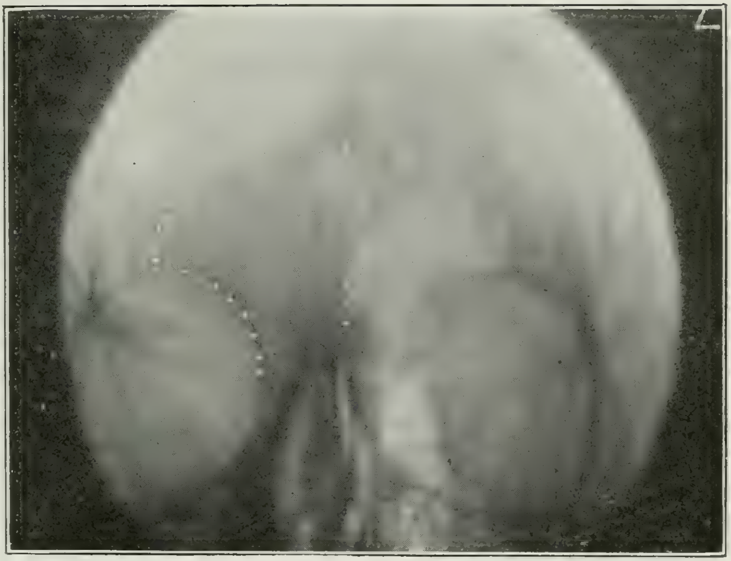

Skiagraph of right frontal sinus and right ethmoid region.

LXII.

A METHOD OF DEMONSTRATING THE SURGICAL ANATOMY OF THE MASTOID BY MODELS.*

By Jesse Wright Downey, Jr., M. D.,

Baltimore.

Variations in the normal anatomy of the temporal bone are so frequent and minute details that might seem of academic interest only are of such vast practical importance that the aural surgeon must constantly visualize a more or less composite anatomic picture that shall include not only the external aspect of the mastoid region but more especially the complicated system of cells that exist within the mastoid and petrous portions and which are, as Sir Charles Ballance so clearly emphasizes, "quite distinct from the medullary, the diploetic and the Haversian spaces proper to the bone substance."

Of course it is most essential that one first acquire his mental impressions of the anatomy of the mastoid and tympanic cavities, in their relationship one to the other, and to contiguous structures by the study of many temporal bones and by the practice of the various surgical procedures upon the cadaver. Knowledge so gained may be, however, of only relative value in the performance of the actual mastoid operation for the cure of disease, for the most constant concomitant of a pathologic process is the change wrought in the appearance, the structure and the relationship of the part involved and, therefore, it is also necessary to be prepared to meet anatomic condition which are the result of the disease itself.

It is these variations in both normal and morbid anatomy which place the modern mastoid operation in a class of surgery requiring not only technical training but the ability to interpret operative findings that follow the exception rather than the rule, and as it is by no means easy to describe one's findings at the time of operation, either in written words or by flat drawings, a clinical history, especially when presented before a class of students or a medical society, can be much enhanced by a model of the operative findings. No operation lends itself better to modeling than does the mastoid operation, and I hope to demonstrate that the method is practical.

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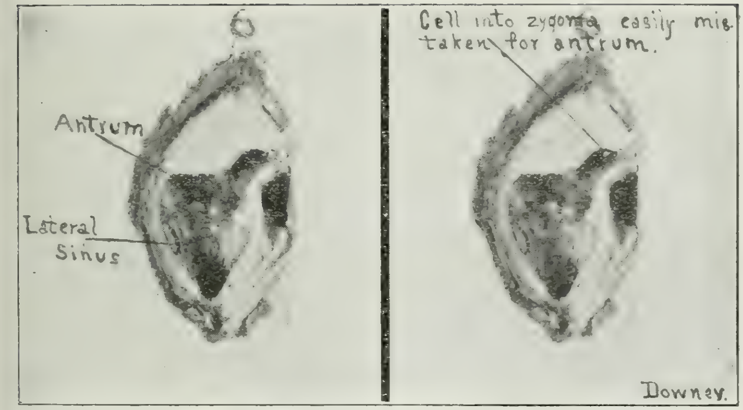
*Presented at the meeting of the Southern Section, American Laryngological, Rhinological and Otological Society, Baltimore, February 4, 1922.

The twelve models which I wish to show have been modeled in artist's water clay from actual operations. Measurements were made at the time of operation, dictated to a nurse, and the model made within a few hours after the operation. Any otologists will find the process surprisingly easy, if he will take a piece of clay and imagine he is operating. The attempt to reproduce the appearance of the mastoid cavity teaches one to pay the strictest attention to details at the time of operation, and certainly good mastoid surgery, like genius, is dependent upon the ability to take infinite pains.

Twelve models of actual operations were exhibited, demonstrating: Extensive destruction of the mastoid process. Anomalous positions of the lateral sinus. Zygomatic cells simulating the antrum. The difficulty of reaching the antrum in certain sclerotic bones The marked alteration in anatomy occasioned by chronic suppurative conditions, etc.



Stereoscopic photograph of one of the models described in this article. This should be viewed with a stereoscope to obtain an idea of the value of the models for teaching purposes.

LXIII.

THE TECHNIC OF THYROTOMY.

By Arthur E. Hertzler, M. D.,

Halstead, Kansas.

There has been a good deal of dispute among laryngologists as to the advisability of the external operation for intrinsic tumors of the larynx. The opponents advance the difficulty of the operation and the disturbance of healing as arguments against the general adoption of this plan in the removal of intralaryngeal tumors.

I have no license to enter into a discussion with specialists in this field, but one need have no hesitancy in pointing out that the more or less blind pulling at the tops of malignant tumors is wholly unsurgical, and, on my own account, I am prepared to assert that the external operation is technically safe and simple, and complications following the operation are negligible. It is to establish this position that this paper is written.

The technic I have adopted is as follows:

A line is infiltrated endermically two and a half inches long, having its midpoint over the height of the Adam's apple (Fig. 1). A 1 per cent novocain solution, containing 10 minims of adrenalin to the ounce of the solution, is used. From this line the subdermal tissues down to the surface of the cartilage is injected. The tissues should not be unduly edematized, because it causes disturbance in the anatomic landmarks.

From this same line the superior laryngeal nerves are blocked. This is done by passing the needle over the surface of the thyroid cartilage to its lateral border, then by pressing on the skin over the end of the needle and at the same time drawing the syringe lateralward the point of the needle can be made to pass into the space, lateral to the thyroid cartilage, in which the nerve lies. If the tip of the thyroid gland extends so high the needle must be made to pass between this and the thyroid cartilage (Fig. 2).

It is now necessary to anesthetize the laryngeal mucosa. This is done by passing the needle obliquely through the cricothyroid membrane and through the thyroid cartilage and depositing the solution between these structures and the mucosa (Fig. 3). This is the only step in the operation that requires any care. The needle is passed so that the bevel edge of the needle will lie parallel with the mucosa when the point appears in the space between the thyroid cartilage and the mucosa. The sense of touch must guide one to this space. Care must be taken lest the needle pass through the mucosa as well. If it does the patient will cough and destroy his confidence in the operator. One or two cc. of the solution should be slowly injected at each of these points. If the solution is injected too rapidly it does not have time to diffuse itself and the patient may resent the sudden increase in pressure. In old patients there may be a deposit of lime salts in the midline of the thyroid cartilage so that the needle cannot be pushed through. In that event some place lateral to the midline must be sought which will permit the passage of the needle.

The size of the needle in the figure (Fig. 3) is exaggerated in order to make the direction of the beveled edge more apparent. In practice a 22 caliber needle is used. The use of a small needle is important, for it is conducive to delicacy of touch in passing it through the structures overlying the mucosa.

This done, the operation may proceed without interruption. An incision is made along the line of primary infiltration. Vessels are looked for, and when exposed are double clamped, cut and tied. The thyroid cartilage and the cricothyroid membrane are exposed (Fig. 4).

These structures are then split exactly in the midline. I prefer to use a sharp knife, cutting first the cartilage and then the mucous membrane. This last named structure is cut in the lower half first. The edges are retracted and the vocal cords are located in order that the mucosa may be severed without injuring them. In old individuals in whom there is extensive lime deposit in the thyroid cartilage, a stout pair of scissors may be used in cutting that part of the structure.

If the use of scissors is required, one must make sure that the blade introduced within the larynx lies accurately between the vocal cords, for when the structure gives way it is likely to be with a jerk and a pop, and if care is not exercised the cord may be injured.

After the mucosa is cut the two parts of the thyroid cartilage are carefully retracted with small retractors. The entire interior of the larynx is then exposed to view, and the needed operation may be proceeded with without exciting reflexes or otherwise disturbing the tranquility of the patient.

Should the operator have been unskillful in his anesthetization and reflexes are excited, local applications with cotton tampons saturated in a cocain solution will supplement the anesthetic.

If the area to be attacked is vascular, the operation will be facilitated by applying tampons saturated with an adrenalin solution.

I prefer to operate as much as possible with the electrocautery, because it is possible in this way to retain a bloodless field during the operation and to insure a complete hemostasis after the operation has been completed.

After the operation has been completed the retractors are removed, allowing the cut edges of the thyroid cartilage to resume their natural position. The fascia covering the cartilage is united in the midline with catgut sutures. No sutures are passed through the cartilage. The skin is closed with interrupted horsehair sutures.

In my earlier operations I placed a tampon over the cricothyroid membrane, keeping the skin separated at this point. The idea was that should obstruction to respiration occur this would furnish a convenient point for the passage of tracheotomy tube. Such an emergency did not arise, however, and I now close the wound completely.

A small gauze pad held by adhesive strips is placed over the wound. The patient is encouraged to sit up at once but is forbidden to talk for a week.

I have had no difficulty in the healing of the wound and no difficulty with any disturbance of respiration during the healing of the wound. Should a tracheal fistula form I should proceed to close it. I have inherited such fistulas from other operators, but never have experienced any difficulty in closing them. I employ the technic commonly used by surgeons in closing vesicovaginal fistulas.

As already stated, I do not wish to enter into any quarrel with laryngologists as to the merit of the external operation as pertains to the management of intralaryngeal lesions, but from my experience neither the difficulty of the technic nor the uncertainty of securing union of the wound can justly be used as an argument against the external operation.

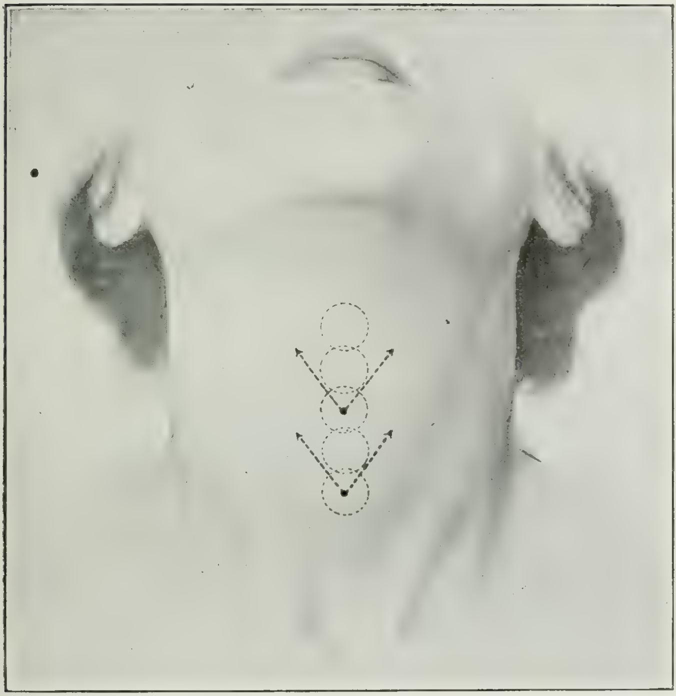


Fig. 1. The circles represent the endermic wheals made by the injection of the solution. The arrowed radiating lines show the direction the needle is passed when the tissues lying between the skin and thyroid tissue is being infiltrated.

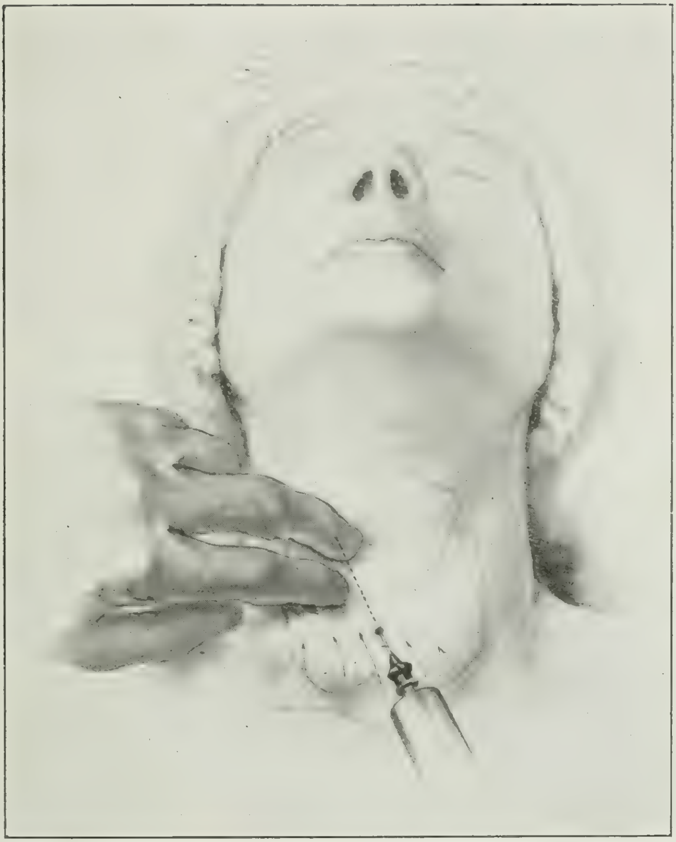


Fig. 2. The finger lying on the skin depresses the point of the needle so that it enters the space between the thyroid cartilage and the tip of the thyroid gland. The superior laryngeal nerve lies on the upper end of this space.

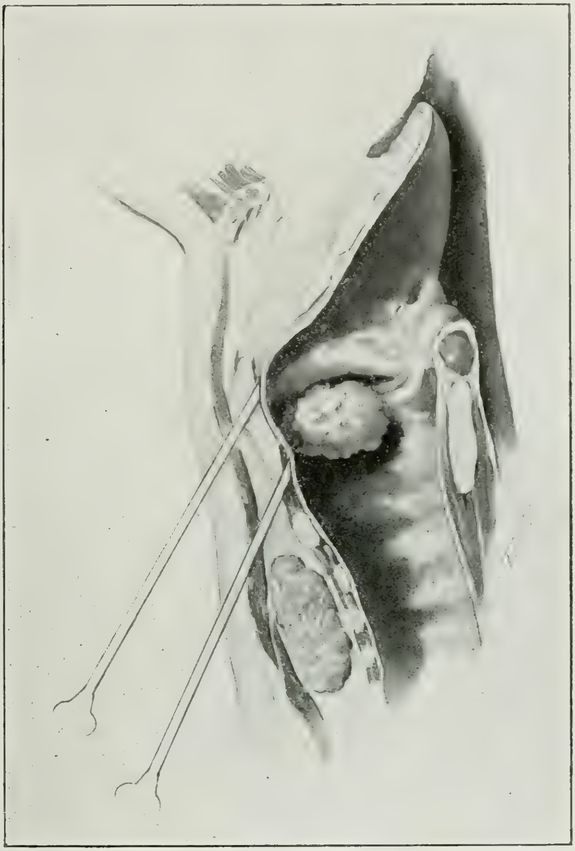


Fig. 3. Schematic diagram of a cross section of a larynx. The needles are passed obliquely through the soft parts and through the cricothyroid membrane and the thyroid cartilage. The tip of the needle is halted the instant it passes through these structures. It then lies between these structures and the laryngeal membrane. The anesthetic fluid is deposited here. Note the beveled edge of the needle lies upon and parallel with the deep surface of the musoca.

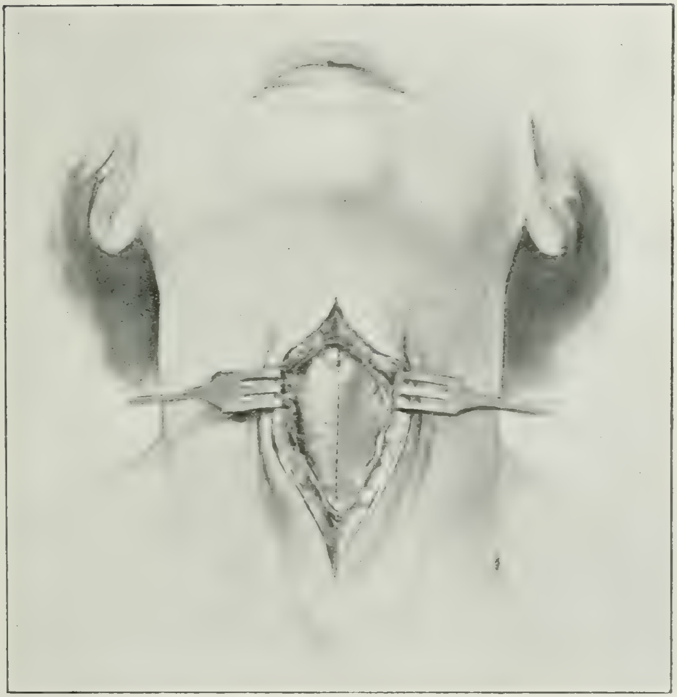


Fig. 4. The soft parts have been severed and are held apart by retractors exposing the cricothyroid membrane and the thyroid cartilage. The dotted line shows the site of the proposed incision.

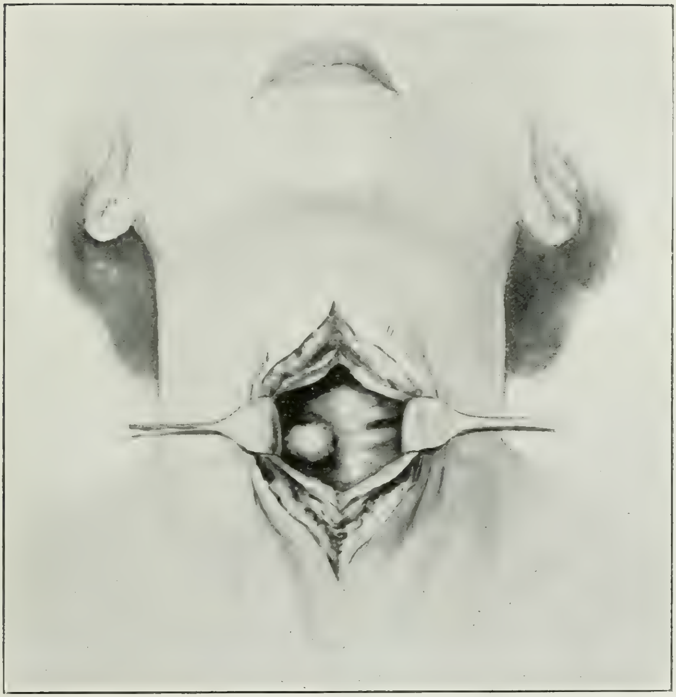


Fig. 5. The cricothyroid membrane, the thyroid cartilage and the laryngeal mucosa have been severed in the midline. The retractors expose the interior of the larynx. The tumor shown is a broad based papilloma which was removed with the electrocautery.

LXIV.

SUBCUTANEOUS EMPHYSEMA OF THE NECK AND CHEST FOLLOWING TONSILLECTOMY IN AN EPILEPTIC. RECOVERY.*

By Sylvan Rosenheim, M. D.,

Baltimore.

FROM THE NOSE AND THROAT DEPARTMENT OF THE HEBREW HOSPITAL,

Osler defines emphysema as the condition in which the infundibular passages and the alveoli are dilated and the alveolar walls atrophied. Surgical emphysema, subcutaneous or traumatic emphysema is that condition in which air or other gases are found in the subcutaneous tissues.

Among the varieties of subcutaneous emphysema are:

Inflammatory or gangrenous emphysema, caused by the gas bacillus of Welch.

Congenital emphysema. Occurs in infants where there is a congenital weakness of the lung. A spell of violent coughing may cause it.

Spontaneous emphysema. May occur after extensive extravasation of blood. It is due to the gas produced by acids, such as lactic, acting on the contained blood.

Traumatic emphysema. This may be of external origin, such as wounds of the face, stab wounds of the neck or chest, etc., or of internal origin from the nose, pharynx, larynx, trachea and lungs, wounds or disease in the gastrointestinal tract or following excessive straining during pregnancy, etc.

There are two ways in which the air may get into the subcutaneous tissues from the lungs: 1. The intrapleural route. The air must pass through the visceral and parietal pleura. In order to do so, adhesions must exist between the pleuræ at this point.

2. The extrapleural route. Air sacs on the periphery of the lung rupture and the air follows the line of least resistance,

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*Read before the Southern Section of the American Laryngological, Rhinological and Otological Association, Baltimore, Feb. 4, 1922.

travels beneath the visceral pleura to the hilum of the lungs, follows the reflection of the pleura on the big vessel and enters the mediastinum and then follows the fascial planes into the tissues. There is in these cases an increased intrapulmonary pressure.

Subcutaneous emphysema following removal of the tonsils is apparently, from a survey of the literature, such a rare occurrence that the following report should be of interest:

S. B., male, aet 21 years, entered the Hebrew Hospital March 11, 1912, under the care of Dr. Irving J. Spear, for thorough examination and treatment for epilepsy. The family history is unimportant. The patient has had measles, chicken pox and mumps. He gives a history of epilepsy, the attacks having occurred at irregular intervals since the age of 1½ years. At the present time he complains of some trouble in hearing.

Besides the presence of hypertrophied tonsils and adenoids the physical examination was of no special interest. The tonsils were removed under ether anesthesia. A weak solution of adrenalin chlorid was first injected around the tonsils and then the tonsils removed by sharp dissection with the knife. It was not necessary to tie any vessels. The adenoids were then removed by curettement. There was considerable trouble during the operation, due to the patient taking a poor anesthetic. When partly asleep he had a slight epileptic attack. The operation lasted one hour, having began at 3 p. m. That evening the patient's condition was not good. He breathed very heavily and frothed at the mouth, expectorating some blood clots. He seemed to struggle for air. His temperature was 103.2, pulse 150. He was not seen at this time by the writer. The next morning an emphysematous swelling was noted in the neck on both sides, extending almost to the clavicle. The breathing was easy and the patient was more comfortable but complained of pain in the neck and abdomen. The stools contained some blood. The leucocyte count was 37,000. The next day the emphysema had extended farther downwards, but the patient's condition was better. The leucocyte count was 26,000. The hemoglobin was 85 per cent. During the next two days the emphysema extended, covering the front of the chest and the axillary region. His general condition commenced to improve. His temperature returned to normal on the tenth day and the emphysema gradually receded. During his stay in the hospital he received hydro- and electrotherapeutic treatment as well as medicinal. He had but one epileptic attack while in the hospital. He was discharged March 20th, much improved.

We have been able to find but one case similar to ours in the literature of this subject. That was reported by Parrish in the Laryngoscope in 1920. It occurred in an adult male following the removal of the tonsils with tonsillotome and punch under ether anesthesia. There was rather free bleeding during the operation and a buttonhole was made in the posterior pillar. Immediately following the operation there was extensive emphysema of the neck and face and anteriorly on the chest. The head and neck were extended, the breathing was rapid and shallow, the pulse 128 and thready. By retaining the mouth open with a gag and flexing the head the condition of the patient was immediately improved. Nothing abnormal was seen in the nose or throat. The air gradually was absorbed, first from the face and neck and later from the chest, and there was a complete recovery.

Woodward reports a case of emphysema of the head and neck, following the perforation of the anterior pillar of the fauces by a pipe stem. Stewart had a case in which there was an extravasation of blood into the subcutaneous tissues of the neck following the removal of the tonsils in a boy, aged seven years. This case ended fatally. The prognosis, of course, depends upon the cause. The cases following influenza and pneumonia usually end fatally. The prognosis in most of the cases is good. In our case, although there was apparently same infection, as indicated by the elevation of temperature and the leucocytosis, the patient appeared at no time very sick. There is no special treatment required for noninflammatory cases. When very extensive some have recommended multiple punctures.

What is to be learned from this case? When one considers the local and general complications that may follow tonsillectomy, such as mutilations of the palate, hemorrhage, local infections of the wound, general sepsis, infections of distant parts of the serous cavities and lungs, it seems that the lesson is quite clear. It is to perform the operation so as to conform to all the rules and regulations of modern surgery. Reading from week to week of the various new instruments devised to remove the faucial tonsil, one wonders if we of this specialty do not pay too much attention to the instrumentation and not enough to the principles involved. According to our ideas, developed through twenty-one years of work in this field, we have come to the following conclusions:

1. That a careful preliminary examination of each patient should be made.

2. When the operation is performed under general anesthesia, ether is the anesthetic of choice. This should be administered only by a specialist in this line, using the warmed ether vapor administered through a tube inserted on the top of the tongue depressor as in the Davis mouth gag.

The operating table should be at an angle of about 25 degrees with the horizontal, the head of course being down, thus preventing aspiration of blood and debris into the trachea and lungs. The precautions will, we think, avoid the complications of ether pneumonia and lung infections.

In adults we prefer the use of local anesthesia, thus avoiding any possible complications from the use of ether as well as the unpleasant ether sickness. The main point in the use of the local anesthetic is to use a very weak solution for injection and allow none to be swallowed by the patient. Usually a hypodermic injection of morphin and atropin is given one-half hour before the operation. I use a one-eighth per cent solution of cocain with four drops of 1/1000 adrenalin chloride solution to the ounce, previously swabbing the throat with a moistened applicator on which powdered cocain is spread. I have been using this method for seventeen years with as yet no untoward results.

3. Hemorrhage, long the bugbear of this operation, has been reduced to a minimum by the procedure to be described. However, when it does occur, pressure is made by one or more sponges held in Kelly clamps, placed in the tonsillar fossæ, and if this does not stop the bleeding, the bleeding point or vessel is grasped with forceps and tied with catgut. We have never found it necessary to use the vacuum apparatus to keep the throat clean, the slight bleeding being readily taken care of by

sponges held on Kelly clamps. Where there is a suspicious history of bleeding previously, it might be well to apply a solution of cephalin, 10 per cent, to the fossæ after removing the tonsils.

4. I am fully persuaded that all operative procedures based on the guillotine principles are reversions to crude tonsillotomy performed years ago. The operation is performed as follows: In ether cases, the space between the capsule of the tonsil and the fascia of the superior constrictor is infiltrated with normal saline solution to which four drops of a 1/1000 solution of adrenalin chloride to each ounce is added. The tonsil is grasped at the upper pole with tonsil forceps and a small incision is made in the plica triangularis. The flat blade of the knife is inserted in this opening, and the dissection of the upper lobe from the supratonsillar fossa is effected, an assistant meanwhile assisting the operator by following the dissecting with a broad retractor. The blunt dissection is continued with a small hard gauze sponge held in a Kelly clamp. When the tonsil is dissected free from the pillars, the operation is completed with the Pierce-Mueller snare. Any bleeding that occurs is controlled as outlined above. If adenoids are present they are then removed and the adenoid bleeding is controlled by a sponge made to fit the nasopharynx. While this pack remains in the nasopharynx a few minutes, the tonsillar fossæ are inspected for bleeding points. The time consumed for the operation averages from fifteen to twenty minutes under ether. When cocain is used the time consumed is usually the same, being dependent mainly upon the idiosyncrasies of the patient.

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LXV.

A CASE SHOWING UNUSUALLY EARLY METASTASIS IN CARCINOMA OF THE VOCAL CORDS.

By Arthur E. Hertzler, M. D.,

Halstead, Kansas.

Not infrequently in operating on a malignant disease, which appears to be in an early stage, we are disappointed by a prompt recurrence. That malignancies are not so early as they seem, or are of a more malignant character than we judge, is the cause of this disappointment. It is not so infrequent, as a matter of fact, that one discovers the metastasis before the original growth is discovered. I have in several instances seen carcinomas in the axilla in patients in whom, even after amputation of the breast, no malignancy area could be found. I recently observed a case of malignancy of the vocal cord marked by the discovery of the metastasis before the intralaryngeal lesion was discovered.

My failure to recognize the importance of a metastasis in the particular gland in question was due to ignorance of the lymphatic drainage of the larynx. Inasmuch as a considerable number of prominent laryngologists choked on the same unsavory morsel causes me to report the case in this place.

It is as follows: Four years ago a prominent colleague had some irritation of the throat. He discovered an enlarged gland in the right side of the neck soon after. He consulted six of his friends and all failed to find more than inflammatory lesion within the larynx and ascribed the enlarged gland to an infection. I examined him at this time and found a hard, smooth gland below the thyroid cartilage and lateral to the common carotid artery. It had a density never attained by an inflammatory gland. There was but the one gland affected. I made a diagnosis of secondary malignancy from a primary tumor to me unknown. I advised removal of the gland and the advice was accepted. Under local anesthesia I did a complete block dissection of that side of the neck from the clavicle to the mastoid process.

As was suspected at the time of physical examination, but one gland was involved. It lay lateral to the jugular vein but upon it at the level of the bone indicated. It was easily separated from the jugular vein when the specimen was examined in the laboratory. The gland measured 1 by 1 by 1.5 cm. and was smooth and firm. On section the small pin point areas characteristic of malignancy could be seen. The slide showed epithelial nests with considerable round celled infiltration (Fig 1). No other evidence of involvement could be found in the remainder of the specimen.

The patient was given X-ray treatment every three months by a competent radiologist. Nothing suggesting recurrence ever appeared in the neck. The patient presented himself every three months for inspection, but nothing was ever detected suggestive in the least of a recurrence. Attempts were made to reassure him, even to the extent of making a diagnosis of primary endothelioma of the gland removed, but nothing availed. He continued to insist that there was something wrong.

Three years after the date of the operation the throat trouble became worse. A laryngologist then diagnosed carcinoma of the right cord. This diagnosis was confirmed by several other equally competent men. So far as I was able to determine, there was a growth the size of a grain of corn involving the ventricle and the posterior end of the right vocal cord. The neck remained free from any sign of recurrence.

The patient consulted a noted laryngologist, who advised the use of radium. This was applied. He died nine days later, it is said, of starvation because of refusal to take nourishment.

The moral to this tale is that it should have been suspected, when the malignancy was discovered in the lymph gland in that particular location, that there must be a hidden malignancy somewhere within the larynx. The lymph gland represents the drainage area of the false cord and ventricle, according to Most (Fig. 2). According to this author, the lymph channels in the false cords are numerous and wide, a condition which makes for early metastasis of malignant growths occurring in this region. Speaking in the abstract, a general surgeon would by instinct have performed an exploratory thyrotomy in order to determine the source of the malignancy. In this instance an early discovery of the primary lesion might have prevented the disastrous outcome. The rate of growth of the primary tumor obviously was not rapid, and diagnosis by laryngoscopy evidently was not exact enough for the requirements of this particular case.

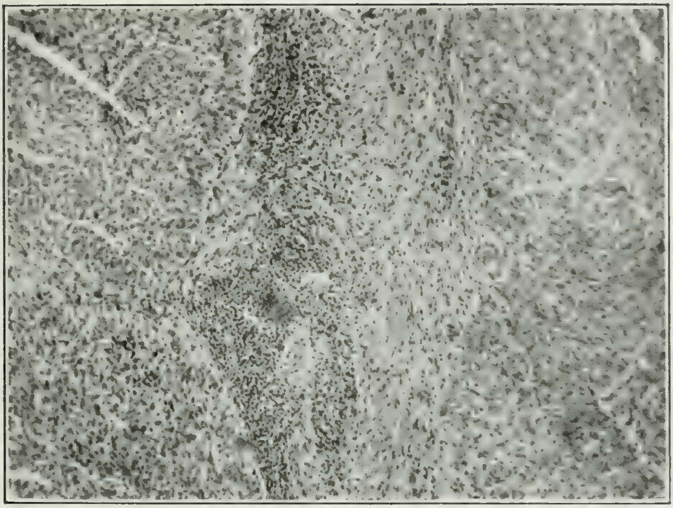

Fig. 1. Low power slide of the lymph gland obtained from the neck.

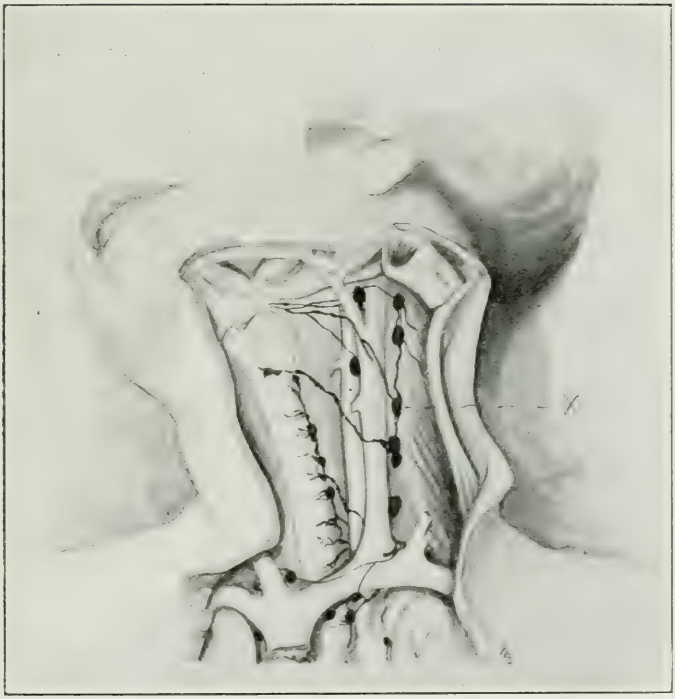

Fig. 2. Lymph drainage of the neck (copied from Most). The gland marked X represents the gland removed from this case.

LXVI.

A FATAL CASE OF VINCENT'S ANGINA.

By David N. Husik, M. D.,

Philadelphia.

Fatal cases following Vincent's angina have been reported by Bruce, Meyer, Halsted and Theisen. The writer would like to add one more case to the above list.

On March 29, 1922, I was asked to see a patient with a sore throat in the ward of the university hospital on the service of Dr. Alfred Stengel. The throat has been sore on one side for two days.

Clinical examination: Right tonsil is covered with a thick, grayish membrane, which extended to the lateral pharyngeal wall, typical of Klebs-Loeffler, with the exception of the odor. Several cultures were negative for diphtheria, but smears were positive for Vincent's.

March 29. Membrane was removed and throat touched up with 25 per cent silver and peroxid, and saline gargles ordered. This treatment was kept up for two days without any results.

March 31. On removing the membrane there was considerable bleeding, the throat was very sore, and the treatment was changed to neoarsphenamin both locally and intravenous. Antiseptic gargles were constantly used.

April 4. Patient felt better, as far as pain was concerned, but the membrane was spreading over the posterior surface of the epiglottis, and the arytenoids were slightly edematous. Temperature 101, very foul odor, and culture again negative for Klebs-Loeffler.

April 3. About the same during the day, except some swelling of epiglottis and impairment of respiration. Very dyspneic during the night. Our treatment seemed to have no effect.

April 4. Epiglottis and arytenoids much more edematous, and only the posterior ends of cords could be seen and their motion was feeble. Was having a good deal of difficulty in breathing. 11 p. m. I was called to the hospital for a tracheotomy, as patient was gasping for breath and was cyanotic. Was perfectly comfortable with the tube and was resting easy. The inner tube was removed every half hour and cleansed and larynx treated locally.

April 5, 6, 7. Patient improving, membrane from throat subsiding, but is weak and takes little nourishment.

April 8, 2 p. m. I was hurriedly called for sudden blockage of tracheotomy tube.

Examination of larynx at this time shows edema greatly subsided, but there was an abductor paralysis of the cords. Dr. Chevalier Jackson, who happened to be in the house, kindly saw the case with me at this time, and he suggested further exploration of the neck, and kindly gave me a long canula, much longer than I had ever used. After the insertion of the long canula there was an excellent supply of air, and the patient was again quiet. On account of the anemic condition 500 cc. of blood from her husband was transfused. At midnight she became unconscious. Intravenous infusion of 200 cc. 3 per cent $NaHCO_3$ at 1 p. m. Tracheotomy tube never became clogged until death, April 9, 6:40 p. m.

History of patient prior to sore throat: M. H., 39, white, married, admitted to the university hospital, March 6, 1922, to the service of Dr. Alfred Stengel, with a diagnosis of secondary anemia and vaginal hemorrhages. The vaginal hemorrhages date from a miscarriage at three and one-half months for which she was treated at several hospitals.

Previous medical history: Diphtheria ten years ago; miscarriage five years ago.

Family history: Father died at 80; mother living and well; husband living and well. Three children living and well; two dead, one at one week and one at one year.

Physical examination: Practically negative except heart. Heart—Apex beat palpable in fifth interspace, just outside the midclavicular line. Blowing systolic murmur at apex, transmitted to axilla, and heard also at base. Second sound high pitched.

Urine: Amber, flocculent sediment, specific gravity 1010, acid reaction, heavy cloud albumen, no sugar. Scope shows: 10-12 narrow epith. and pus L. P. F., occasional narrow dark granular, and occasional narrow granular casts, R. B. C. 1—3 H. P. F., W. B. C. 100—200 H. P. F.; few epith, no crystals.

Simple gastric: Contents, 130 cc. watery, gran, sediment, acid, white; total acidity, 50; free H. C. L., 20; lactic acid trace, occult blood very slight trace. Starch, occasional yeast; fat, +; other food masses, 0; R. B. C., 0; W. B. C., 0; epith., 0; mucus, 0; Oppler Boas bac., 0; Sarcina, 0.

Blood: Hb. 32 per cent; R. B. C., 2,300,000. Polchrom, 0; anis, slight, very slight poik, nucleated reds 0; basic degeneration, 0. Patient type. Blood differentials: Neut., 74; lymph., 16; L. M., 8; Trans., 2; Eos., 0; Bas., 0. Wassermann negative.

Tentative diagnosis: Secondary anemia; chronic nephritis.

Autopsy: Dr. McCutcheon.

Body of white adult female. Autopsy 15 hours after death; normal framework, moderately emaciated, measuring about 160 oms., weighing about 110 pounds. Rigor and liver well developed. Skin shows no edema, jaundice or eruption. No glandular enlargement is noted. On the anterior aspect of neck is a midline incision about 9 oms. in length, connecting with the trachea. The exposed tissues have a blackish discoloration. No suppuration is present. Scleræ are clear. Orifices negative, except for atrophy of the alveolar processes in the mouth. Hair distribution normal; in the right forearm is a recent surgical incision (transfusion). Trunk and genitals normal.

Internal Examination.—There is a single adhesion at the apex of the right lung; otherwise the pleural cavities are normal. Pericardial sac contains little fluid. Aorta of normal caliber; possesses a smooth intima. Tongue shows no changes.

Pharynx—Lower end of pharynx examined shows a blackish discoloration, a small quantity of black, friable, false membrane adherent to the mucosa. The same type of membrane, but non-adherent, is found in the larynx, trachea and right bronchus, extending into the secondary bronchus, but no further. The exudate is nowhere very abundant, and does not appear to occlude the air passages. Left bronchus contains no exudate. The true vocal cords are pale and swollen. Epiglottis, trachea and bronchi are congested. The trachea shows two longitudinal incisions, one of these, the upper, passing through the median lobe of the thyroid gland; the latter is normal.

Lungs—Upper lobe shows no areas of consolidation, and no apical scars. Lower lobe contains a number of consolidated lobules, gray or red. Same condition is found in the upper and lower lobes of the right lung, together with a moderate amount of fluid and redness. Middle lobe is inflated and pale.

Abdomen.—Peritoneum smooth and glistening; no fluid or adhesions. The diaphragm reaches to the third interspace on the right side, fourth rib on the left. Appendix free.

Esophagus, stomach, gall bladder and intestines examined and found normal.

Liver—1400 grams; flaccid, lower border rounded, capsule unthickened, cut surface pale, yellow, lobulation, and central veins indistinct. Central part of lobules appears to have a yellow color, texture friable. Two nodules are met with about 1 cm. in diameter, one in the subcapsular position, well circumscribed, and bulging, and of the same color, and consistency of the liver tissue.

Left Kidney—140 grams; flaccid capsule, strips easily, leaving a smooth or finely granular yellowish surface with distinct stellate veins. Cut surface shows a striking yellow color of the inner half of the cortex. Cortical markings indistinct. Medulla appears normal. Right resembles fellow.

Uterus.—Cervix is eroded, and that part of it which should protrude into the vagina is missing. There appears to be a slight longitudinal tear. The lumen of the uterus is filled with a soft new growth, yellowish and hemorrhagic, adherent in some places to the uterine wall, and apparently invading the latter near the fundus. The fundus is enlarged; in consequence this growth extends down into the cervical canal, where it presents a blackish discoloration, and is friable.

Histologic protocol—Sections were prepared and examined histologically from the following parts: Heart, lung, spleen, liver, kidney, uterus, pharynx and thyroid gland.

Pharynx—Mucosa is necrotic and is represented by a fibrillar eosin staining material, in which bacteria can be seen. The submucosa is thickened by a fibrinous exudate with small areas of hemorrhage. The cells are both polynuclear and mononuclear, in about equal numbers. There is a proliferation of connective tissue with growth of capillaries into the necrotic mucosa. Lymph follicles are only slightly altered. The voluntary muscle on the mucosal side has been largely destroyed, the remaining fibers being hyalin. In this zone is found a fibrillar material apparently composed of varying elements—fibrin, connective tissue, and detritus of muscle cells. The next zone shows the muscle fairly intact, except for frequent examples of hyalin degeneration; there is a marked overgrowth of connective tissue of a cellular type, and occasional suggestions of muscle regeneration. The outermost zone shows normal muscle.

Thyroid Gland—Interstitial tissue shows slight increase in some areas, but there are no exudative changes. Acini are of normal size, filled with colloid.

Balteriologic Examination—Dr. Bothe.

Culture of Heart Blood—Staphylococcus aureus.

Culture of Trachea—Staphylococcus aureus (predominating); micrococcus catarrhalis; nonhemolytic streptococcus.

Smears from Trachea and Lung—Gram positive cocci predominating; few streptococci; gram negative diplococcus.

LXVII.

X-RAY TREATMENT OF TONSILLAR AND LYMPHOID TISSUE.

By Major John H. Trinder, M. C., U. S. Army,
Station Hospital, Fort Sam Houston, Texas.

The earliest record of the effects of the X-rays on lymphoid tissue was made by Heinicke in 1905. Since then various workers have noted the susceptibility of this tissue to X-ray dosage, of such strength as to have no appreciable effect upon normal tissues. This has had its practical application in the X-ray treatment of such conditions as Hodgkin's disease, tuberculous cervical glands, various skin lesions and the leukemias. Within the last three years Dr. W. D. Witherbee of New York has done considerable commendable work along this line. In 1919 he began treating a series of cases of hypertrophied tonsils and other lymphoid structures of the nasopharynx, making careful clinical and bacteriologic observations, and recording the same in various medical journals. To date he has handled over 500 cases; and his conclusions are definite as to the efficacy of this method of treatment, and have been borne out in most part by the series upon which this paper is based, although there is one particular reaction which he obtained, but which was not observed as consistently as he states. This will be mentioned later, with a probable explanation of the different results. Associated with Dr. Witherbee in his work were Doctors Murphy, Craig, Hussey and Sturm. Dr. William G. Hermann of Asbury Park, N. J., and Dr. H. W. Van Allen of Springfield, Mass., have also contributed to the literature on this subject. The work of the above mentioned gentlemen furnished the impetus for this present series, and their writings are freely referred to and at times quoted in this paper.

The Faucial Tonsils (Palatine Tonsils, Amygdalæ).—The tonsils consist of a mass of lymphoid tissue, which projects slightly from each side into the cavity of fauces and are covered by a layer of stratified epithelium continuous with that which lines the oral and pharyngeal cavities. The lymph nodules which compose the tonsil immediately underlie the epithelial coat, and are imbedded in the areolar connective tissue. The epithelial coat here and there penetrates the substance of the organ in the form of invaginated funnel shaped depressions, the crypts or follicles of the tonsil. The direction of the crypts in the upper third of the organ is downward and outward (C. P. Johnson). The ducts of many mucous glands open into the connective tissue which surrounds the tonsil on all but its faucial surface. The crypts are lined throughout by a layer of stratified epithelium, which is continuous with that on the free surface of the tonsil, and which becomes progressively thinner as it recedes into the deeper recesses of the crypts.

Many of the lymph cells migrate into the intercellular spaces of the epithelial layer, and even penetrate to the free surface, and thus find their way into the oral cavity, where they are found in large numbers in the saliva as "salivary corpuscles." If such salivary corpuscles are examined in a drop of saliva, freshly prepared, the fine intracellular granules of the polymorphonuclear leucocytes will be seen to undergo an active dancing movement, Brownian motion. The salivary corpuscles are derived not only from the faucial tonsils, but also from other lymphoid tissue which is in relation with the oral mucous membrane—e. g., the lingual and pharyngeal tonsils. The passage of leucocytes through the epithelial surface of the faucial tonsil is so very active that at times the epithelium becomes completely filled with these cells, and it is then difficult to distinguish it from the adenoid tissue beneath. The normal tonsils atrophy after puberty, in contradistinction to the diseased organs.

The Lingual Tonsil.—A collection of lymph nodules is found at the base of the tongue in the median line, between the circumvallate papillæ and the epiglottis. This, because of its similarity in appearance and in structure to the faucial tonsil, is called the lingual tonsil. In this latter, however, the nodules are grouped about a single wide mouthed crypt, the foramen caecum lingui. This crypt is frequently branched and into it many mucous glands of the neighboring lingual mucosa pour their secretion.

The Pharyngeal Tonsil.—The posterior wall of the nasopharynx is supplied with a similar accumulation of lymph nodules, the pharyngeal tonsil. It lies in the median line, and extends downward between the orifices of the eustachian tubes for a distance of 3 cm. (Klein). It contains a considerable number of lymph nodules and several small crypts. The lateral extensions in the vicinity of the tubal orifices are sometimes known as the "tubal tonsils." The pharyngeal tonsil is prone to hypertrophy in youth, in which case it forms the adenoid growths which are so common in strumous children.

Viewing the several tonsils and the associated lymphoid tissue as a whole, it will be perceived that they constitute a lymphoid ring at the gateway to the alimentary tract and also the respiratory. The function of the lymphoid tissue is to produce phagocytic leucocytes for the protection of the body against bacteria and other noxious products. The tonsillar crypts offer favorable foci for the lodgment, invasion and attack of such harmful elements. The location of this annular mass of lymphoid tissue is significant, and it is placed where it can apparently best perform a necessary function. When called upon to increase its functional activity lymphoid tissue responds by hypertrophy; this of itself causes inconvenience by obstructing the channels employed in respiration and phonation. But when unable to respond adequately and thus successfully cope with the infecting agency, the tonsils become diseased. This is commonly considered a cause for removal of the involved lymphoid masses. But it would seem that the excision of the tonsils would result in handicaping the organism in its perpetual combat with bacteria by depriving it of a means of defense. Moreover, in the case of the removal of the faucial tonsils, proper phonation may also be interfered with, although if injury to the pillars and soft palate be avoided in operating this latter risk is minimized. If the X-ray will remove the excess lymphoid tissue from diseased tonsils and restore them in whole or part to their normal state, so that their function as guardians of the human economy, rather than storehouses for infection, can be resumed, then its use will be a decided advance in conservative surgery and preventive medicine.

The principle upon which this method is based might be stated as follows: Both lymphatic and embryonic tissue are more easily destroyed by the X-ray than any other living cell. The tonsil is made up mainly of lymph tissue, as shown above, and the small fibrous tonsil so commonly associated with rheumatism contains lymph follicles in which 90 per cent of the follicle is embryonic tissue, as evidenced by the mitotic figures. The large lymph tonsil is made up of about 40 per cent of embryonic tissue, and the remainder of the follicles engorged with mature lymphocytes. Therefore it is possible to use very small doses of X-ray to promote absorption of the lymphatic element, and which will in no way interfere with any of the surrounding and adjacent cells or glands. From the standpoint of infection, the shrinkage of the tonsil and the lymph tissue of the lateral and posterior wall of the nasopharynx by X-ray, will relieve the distortion of the crypts throughout the entire mucous membrane and thus promote drainage. This is impossible by any known operative procedure. In a series of 52 cases, cultures from the crypts showed the presence of members of the hemolytic in 30 cases, the streptococcus viridans in three and members of the pneumococcus group in 25. After treatment with the X-ray all but four of the first group, all of the second and all but three of the third group had disappeared. As the effect of the X-ray on bacteria is nil, this disappearance must have been due to drainage. This is further borne out by the fact that clinically the crypts appeared to be open and in some cases even gaping.

The technic used by the Rockefeller Institute Board is as follows: The patient is placed with the head tilted so that the axes of the X-ray pass under the angle of the jaw into the region of the tonsils, as in making a radiograph of the lower molars, the patient of course being prone. An exposed area about three inches square on each side of the neck is treated, the face and other parts of the head being protected with heavy sheet lead. The dosage is regulated by a 7 inch spark gap, 5 milliamperes, 10 inch skin distance, filtered through 3 mm. of aluminum, making a dose of from 1 to 1¾ skin units. Time of exposure, 3 to 7 minutes, being less in the child than in the adult. Treatment repeated at first every month for five treatments. This was later changed to every week or ten days for eight treatments. Each treatment was controlled by a preliminary bacteriologic examination of the tonsils, the cultures being taken from the crypts. Treatments are not given if there are signs or symptoms of an acute inflammatory condition of the parts. To quote from Pillsbury of the Army Medical School: This technic will reduce lymphoid tissue with practical certainty; it will never cause a burn, and will only rarely cause swelling of the parotid. I have not known it to cause dryness of the throat, nor has anyone I have talked to found it. If the tonsil is enlarged because of the lymphoid tissue in it, it will shrink down amazingly. If the increase is due to fibrous tissue it will not. Having reduced the tonsil, you have reduced it to its original condition, as it was before infection. The rayed tonsil may now be reinfected and return to its former size." For the treatment of adenoids a similar sized area, 3 inches square, just below the posterior occipital region, with the head tilted forward, is subjected to the same dosage.

The technic used in the present series, at the Roentgenological Laboratory, Station Hospital, Fort Sam Houston, Texas, was as follows: Five inch spark gap, 5 milliamperes, 2 millimeters of aluminum filter; 8 inch skin distance, 3 minutes' exposure to each tonsil, one week interval between treatments, from five to ten treatments, depending upon the bacteria found, being given. Treatments were repeated in all cases until members of the hemolytic group, the pneumococcus group or other pathogenic organism found to be present had disappeared. If no virulent bacteria were found, a minimum of five treatments were given. The above technic differs from Witherbee's in the following respects:

1. Spark gap reduced from 7 to 5 inches.
2. Filter reduced from 3 to 2 mm.
3. Skin distance reduced from 10 to 8 inches.
4. Intervals between doses made definitely one week in all cases.

This rearrangement of the factors was made by Major J. D. Mingos, roentgenologist at the hospital at that time, with the idea of arriving at the smallest dosage compatible with greatest clinical results. It will be noted that while the spark gap was reduced there was also a reduction in the thickness of the aluminum filter and in the skin distance. The interval between treatments was also reduced to the shortest deemed safe.

Of the 46 cases recorded at the Rockefeller Institute, all but four showed marked atrophy of the tonsils and lymphoid tissue, with an opening of the crypts, and drainage apparently reestablished. The surface of the tonsils became smooth and normal in appearance, and the invading organisms disappeared usually within four weeks of the treatment.

In the series upon which this paper is based the following number and types of cases were handled:

a. Pharyngitis, in which the tonsils had been removed, some by the writer and the remainder by other operators. In all cases the tonsillar fossæ were free of lymphoid tissue and showing the usual amount of scar tissue. There were varying sized, elevated, red patches on the posterior pharyngeal wall, and in a number there was a distinct vertical red elevated ridge just behind the posterior tonsillar pillar on each side. They all presented symptoms of soreness of the throat, accumulation of mucus, and some had focal symptoms, as pain in the occipital region, back, rheumatic pains, neuritis and a general feeling of being out of sorts. Most of them had had all sorts of treatments by different men, and had finally become discouraged or had been discharged with the diagnosis of "neurasthenia."

It is in this class of cases in which the original operative procedure had not relieved the patient, and no further operation is possible, and which do not react to local or constitutional remedies, that X-ray therapy is a decided boon. All cases cleared promptly, the focal symptoms disappearing, the pharyngeal mucosa became smoothed out and assumed a healthy gray appearance.

Number of above cases treated, 11.

b. Tonsillitis, chronic follicular, in which for various reasons, either constitutional and not good operative risks or because of fear of the operation, or refusal to have it done. All had history of recurrent attacks of sore throat and some had focal symptoms—10.

c. Tonsillitis and pharyngitis, in which tonsils had not been removed for reasons stated under b, and presenting symptoms cited under a.—20.

d. Tonsillitis, chronic follicular, in which all gave history of recurrent attacks of tonsillitis, some having focal infections. In this class a diagram was first made of the throat and a description of the pathology present. One tonsil, that showing the least pathologic change, and adenoids, if present, were removed, under local anesthesia in most cases, a few under general. The removed organ was sent to the laboratory, where it was carefully measured, fixed and sectioned. The remaining tonsil was then subjected to X-ray therapy, using the dosage as given above, and the number of treatments being controlled by the bacteriologic findings. The minimum number of treatments was five, and a few received eight and one ten. At the end of a six months' period, or as soon thereafter as the patient could have the operation, the remaining tonsil was removed and sent to the laboratory for comparative study.—35. Total, 76.

To date the bacteriologic work has been completed on 52 of the above mentioned cases and the comparative histologic study on 16. The remaining cases have not completed the treatments, or the six months' period for the removal of the second tonsil has not passed. The conclusions and observations of this paper are based on completed cases only.

In all cases a careful history was taken, noting the presence of arthritis, neuritis or other manifestations of focal infection. A preliminary culture was taken from the crypts or pharyngeal wall at the beginning of each course of treatment and a culture before each treatment. In this way the number of exposures was controlled and a check kept upon the change in the bacterial flora. All cases received five exposures, and those showing any pathogenic bacteria after the fifth treatment received further treatments until the offending organism had disappeared. Some cases had as many as eight exposures and one case received ten.

The results of X-ray therapy in this series of cases has been very satisfactory from the clinical and bacteriologic standpoint. All cases, with two exceptions, had marked relief of symptoms, both local and focal, and went through a fairly severe winter without the usual attack of sore throat. The improvement was especially noted in the chronic pharyngitis cases, in two of which the process had involved the orifice of the eustachian tubes, producing some deafness and an annoying tinnitus. The hearing in these cases was improved and the tinnitus relieved. In two of the one tonsil cases there was a unilateral otitis media, and the tonsil of the side of the nonaffected ear was removed. After treating the tonsil of the side with the discharging ear the discharge ceased and there was an apparent improvement in the otitic condition, and to date the otorrhea has not returned. One case of badly infected tonsils, in which the left only had been removed, developed a severe attack of tonsillitis of the follicular type, in the remaining organ, after the fourth treatment. He ran a fairly severe course for seven days, but without the marked constitutional effects of previous attacks. After all acute symptoms had subsided the fifth treatment was given. None of the hemolytic or pneumococcic group had been obtained in culture in this case until after this acute exacerbation, when the hemolytic streptococcus was found, just prior to the fifth exposure. In another culture taken eight days later, this organism had disappeared and the tonsil was removed. Another case of a child about six years old, the daughter of a medical officer, developed an acute tonsillitis nine weeks after the last X-ray treatment. In the afternoon she had been playing in the rain in her bathing suit, and that evening complained of soreness of the throat and her temperature went up to 101. The tonsils were red but the follicles were not involved. She reacted promptly to treatment, which consisted only of purgation and rest in bed, and the following day all symptoms had subsided and she was up and about. This case illustrates a particularly interesting phase of the results of X-ray therapy. This child had been subject to frequent and severe attacks of tonsillitis of the follicular type, but the above mentioned attack, coming on after rather marked predisposing factors was very mild, presenting a far different picture from the previous attacks.

The bacteriologic work on these cases was done by Captain Brooks Grant, M. C., and Captain R. L. Holt, M. C., of the

Eighth Corps Area Laboratory, Fort Sam Houston. Captain Holt reports as follows: "This paper takes into consideration fifty-two cases which have had five or more treatments with X-ray, the cultures being taken weekly, prior to their treatment with the rays.

The cultures were taken in the ordinary way with sterile cotton swabs and transferred to standard blood agar plates. They were then incubated for twelve to eighteen hours at 37.5 degrees C. and read. The pneumococci were not typed, but considered as a whole, and the hemolytic group was likewise considered.

Thirty cases showed hemolytic colonies at some time during treatment.

Twenty-two cases showed no hemolytic colonies any time.

Twenty-five cases showed pneumococcic colonies at some time during treatment.

Twenty-seven cases showed no pneumococci at any time.

Fourteen cases showed both hemolytic and pneumococcic colonies at some time.

Thirty-two cases did not show the two combined.

Twelve cases showed hemolytic colonies in the first culture.

Ten cases showed hemolytic colonies in the second culture.

Ten cases showed hemolytic colonies in the third culture.

Thirteen cases showed hemolytic colonies in the fourth culture.

Seven cases showed hemolytic colonies in the fifth culture.

Seven cases showed hemolytic colonies in the sixth culture.

One case showed hemolytic colonies in the seventh culture.

Six cases showed pneumococcic colonies in the first culture.

Six cases showed pneumococcic colonies in the second culture.

Seven cases showed pneumococcic colonies in the third culture.

Twelve cases showed pneumococcic colonies in the fourth culture.

Four cases showed pneumococcic colonies in the fifth culture.

No case showed pneumococcic colonies in the sixth culture.

One case showed streptococcus viridans in the third culture.

Two cases showed streptococcus viridans in the fourth culture.

In many cases showing hemolytic and pneumococcic colonies it was seen that these organisms were present in the first or second culture and would then disappear permanently. In others they would not appear until the third or fourth culture and would then disappear. In still others the colonies would be found in both the above cultures. The natural deduction from this was that possibly the organisms were on the surface in the case of those appearing in the first or second culture and buried in the crypts in cases where they appeared in the later cultures. In the cases where they appeared in the second and fourth cultures, it would seem that possibly they were both on the surface and in the depths of the crypts.

Since the action of the X-ray seems to have no deleterious effects on the organisms under discussion, it would seem reasonable to assume that the disappearance of the colonies was probably due to the alteration in the tissues.

Several cases have been cultured several months after disappearance of the organisms and they remained persistently negative.

To quote Witherbee: "The direct effect of X-ray on bacteria when applied to a growing culture is practically nil. In the series of thirty-six cases treated at the Rockefeller Institute thirty-two were negative to pathogenic bacteria four weeks after treatment, and no change was noticed in the number of colonies of the ordinary bacteria usually found on the mucous surface. Whether these results on pathogenic bacteria are due to the evacuation of the crypts or to an increased local phagocytosis, we are at present unable to state. These results seem to coincide with those obtained in acne vulgaris. In this condition we have a chronically infected sebaceous duct and gland corresponding to the infected crypt and follicle of the tonsil. Acne vulgaris presents one of the most striking and permanent results in X-ray therapy."

Captain H. R. Livesay, M. C., did the pathologic work on this series, but has rendered a report on sixteen cases only. Of the remainder, some are still under study, and the others still in situ in the throats, the six months' period for their removal not having expired. His findings are as follows:

First tonsil, not subjected to X-ray.	Second tonsil, from same case, full course of X-ray.
SIZE.	
1—3.5x2.5 cm.	3.5x2.5x1.5 cm.
2—1.5x1.5 cm.	2.5x2 x2 cm.
3—three pieces, not measured	Three pieces 2.5x2x1.5 cm. 1x1x.8 cm.
4—2.5x1.5 cm.	2.5x1.5x1.5 cm.
5—3.5x2.5 cm.	3 x2.5x1.5 cm.
6—3 x2.5 cm.	3.5x2.8x1.2 cm.
7—2.5x2 cm.	2.5x2.5x1.5 cm.
8—2.5x2 cm.	2 x2 x1.5 cm.
9—3 x2.5 cm.	3.5x2.5x2 cm.
10—3 x3 cm.	3.5x3 x2 cm.
11—3 x2.5 cm.	2.5x2 x2 cm.
12—2 x1.5 cm.	3.5x2 x2 cm.
13—3.5x2.5x1.5 cm.	3 x2.5x2 cm.
14—2.5x2 x1.5 cm.	2.5x2 x1.5 cm.
15—3 x2.5x2 cm.	3 x2 x1.5 cm.
16—Two ragged pieces, not measured	Two pieces 2.5x2x1.5 cm. 2x1.5x1.5 cm.

The measurements were based on the length, the width or distance between the pillars when the tonsil was in situ and the thickness or depth from the mucous surface to the capsule. The last measurement was not taken in the first specimen, as this was thought to be subject to the least variation, and later observation confirmed this view.

Four cases reduced in size, as compared with its fellow.

Five cases, no change in size.

Five cases, larger than its fellow.

Two cases, not comparative tabulation because of fragmentation.

From microscopic examination of thirteen cases of tonsils, before and after X-ray treatment, it is evident that there was no increase of fibrosis, but some vacuolation of the cells of the mucosa, with an apparent widening of the crypts. The lymph follicles show no reduction in size, but in a few cases there appears to be a thinning out of the number of cells. There is no apparent reduction in the amount of lymphoid tissue.

The pathologic and histologic findings do not bear out the clinical observation as shown in the tonsil in situ after treatment, as illustrated in the drawings, and as claimed by other observers, that there is an atrophy of the tonsil. There is one factor to consider in the beginning, and that is as to the actual comparative size of the two tonsils in situ, before any treatments were given. It is in the realm of improbability to hold that any twin organs of the body are exactly the same size. Macroscopically they may appear to be, but when measured in centimeters the difference is apparent.

In observing these cases over a period of six months or more, during the treatment and after it, there was a very perceptible reduction in the size of tonsils subjected to X-ray therapy. In the cases of the markedly hypertrophied organs there was an apparent reduction of one-third to one-half, and in the case of the smaller tonsils they disappeared behind the pillars. But when it came to removing the tonsils which had been given the full course of X-ray treatment, it was found that there was a retraction of the organs into the fossæ and behind the pillars rather than an actual decrease in size. It was also noted that they were more densely adherent than had been the organ first removed and which had had no X-ray therapy. There appeared to be more fibrous tissue in the capsule, as if a peritonsillitis had been active, and there was decidedly less hemorrhage of an oozing character, and the bed of the fossæ was white and often glistening in appearance.

Just why this retraction within the fossa rather than a reduction in the size of the tonsil takes place, it is not possible to definitely state from the small number of cases run through the laboratory examination to date and makes this claim of other observers an open question. The normal tonsil is not a distorted, hypertrophied organ projecting into the oral cavity but lies in its fossa, protected by pillars. Now why should not a swollen diseased tonsil which as a result of X-ray therapy has been relieved of its retained secretions and concretions by the opening and drainage of the crypts and again rendered as near a normal organ as the permanent pathologic change will permit, likewise assume its normal position in the oral cavity? With the above mentioned drainage, is there a shrinkage of the mucosa, with a corresponding retraction of the organ? Or is there a fibrosis of the capsule, pulling in and anchoring the tonsil deep in the fossa? From the observation made upon operating upon X-rayed tonsils, this is a probable explanation. Does the X-ray stimulate the formation of fibrous tissue, through the agency of a peritonsillitis of a low grade assisting in the natural process of walling off infection and a retraction of the organ as a consequence? These and other questions will have to be studied before final and definite conclusions are reached.

It is too early to draw definite conclusions as to the real efficacy of the X-ray in the treatment of throat conditions or as to the permanency of the results. Even with an apparent cure of the case, the tonsils are still present and liable to reinfection, as shown in two cases of this series. I do not believe that X-ray therapy will ever become the method par excellence, to the exclusion of operative measures, for diseases of the throat. While the operation of tonsillectomy is and should always be considered a major one, the present day technic of careful preliminary study of cases, including a coagulation time of the blood, a study of the heart, lungs and kidneys, as in any other major operation, and absolute control of hemorrhage at the time of operation, using ligatures on all doubtful bleeding points, and the use of the suction apparatus in cases of general anesthesia, thus minimizing the danger of insufflating material into the lungs, and not operating in the presence of acute inflammatory conditions, makes this a comparatively safe and sane procedure, and the beneficial results fully justify it, especially in children in whom no contraindications to operation exist.

On the other hand, it certainly has a large field of usefulness in cases which are judged to be poor operative risks and in whom none but emergency operation in the attempt to save life would be considered. Such would be advanced cases of cardiac or pulmonary lesions and Bright's disease and any constitutional condition which would militate against the giving of a general or local anesthesia. It is indicated in the highly neurotic individual and in that unfortunate class who refuse any operative procedure whatsoever. It will be a special boon in cases of hemophilia, with marked tonsillar disease and secondary focal infections therefrom. It will also be the method of choice in singers and elocutionists, in whom the risk of changing the contour of the structures of the mouth and throat, affecting the acoustic properties of the oral cavity and changing the tone of the voice, exists in operative measures.

The objections so far encountered to the X-ray method have been, first, the dangers of a burn. This is impossible if the technic prescribed is carried out. The possibility of injury to the parotid, the thyroid, the pituitary and other adjacent glands has been amply tested in the past ten years in which tuberculous glands of the neck have been treated by much larger doses and more frequency of application, some patients receiving as high as forty exposures, whereas the dose for tonsils and adenoids has never exceeded fourteen treatments in any given case in a series of five hundred cases which Witherbee has treated in the past two years.

In conclusion, I would say that X-ray therapy has certainly given us a very valuable adjunct to the present day methods in treatment of diseases of the throat and should be considered in approaching the treatment of every case and used where operative measures are contraindicated or will not be submitted to by the patient.

In this work I am indebted to Major Wilson C. von Kessler, M. C., and Major J. D. Mingos, M. C., roentgenologists at the Station Hospital, Fort Sam Houston, Texas, for their hearty cooperation; to Captain R. L. Holt, M. C., and Captain Brooks Grant, M. C., of the Laboratory of the Eighth Corps Area, who did the bacteriologic work; to Captain H. R. Livesay, M. C., of the same Laboratory, who did the histopathologic work; to Captain Joseph W. Loef, F. A., who prepared the original chart and diagrams from which the drawings were made, and to First Lieutenant Francois H. K. Reynolds, V. C., Laboratory, Eighth Corps Area, who rendered the finished drawings and chart.

HEMOLYTIC GROUP.

						Treatments				
Case No.	1	2	3	4	5	6	7	8	9	10
1	....	....	....	....	....					
2	....	....	....	....	....					
3	....	....	+	....	....					
4	+	....	....	....	....					
5	....	....	....	....	+					
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7	....	....	....	+	....	+				
8	+	....	....	....	s.v.	....	....			
9	....	....	+	....	....					
10	....	....	....							
11	+	+	....	....	....					
12	+	....	....	....	....					
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17	+	+	+	+	+					
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19	+	....	+	+	....					
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23	....	....	....	+	+	+	....	....		
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32	....	....	....	....	....	....				
33	....	....	+	+	....	....				
34	....	....	....	....	....					
35	....	+	+	+	....	....				
36	+	+	....	....	+	....	....			
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42	....	....	....	....	....					
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PNEUMO GROUPS

Case No.	Treatments									
	1	2	3	4	5	6	7	8	9	10
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52	----	----	----	+	----					

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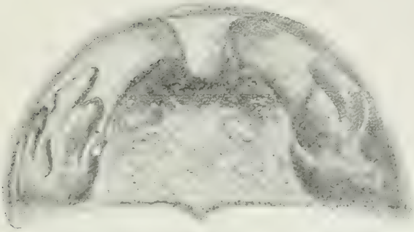
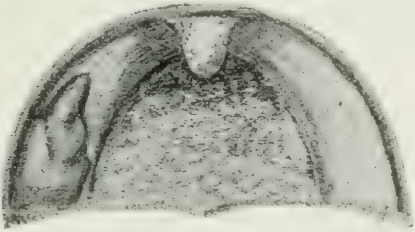
Fig. 1—Case J. N.



a.

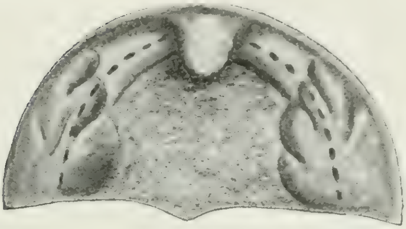
Tonsils enlarged, follicular. History of repeated attacks of tonsillitis. Right tonsil had deep infected crypts on lingual surface. Left tonsil removed, under local anesthesia, Sept. 1, 1921; and sent to laboratory for examination. Right tonsil received six X-ray treatments. Drawing shows throat before treatments.



b.

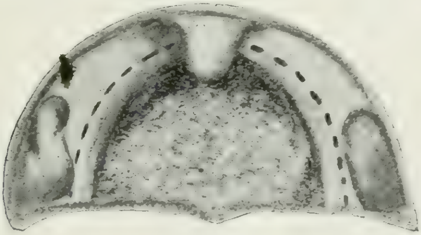
Case J. N.: Appearance of throat after six X-ray treatments. Right tonsil reduced about one-fourth inch in size. Between the fourth and fifth treatments, this case developed an acute follicular condition of the remaining tonsil, with moderately severe constitutional symptoms. (See text.) The reduction in size of this tonsil was only apparent, as upon subsequent removal, it was found to be retracted behind the anterior pillar, and buried deeply in the fossa. It was as large as its untreated fellow; and densely adherent.

Fig. 2—Case H. L.



a.

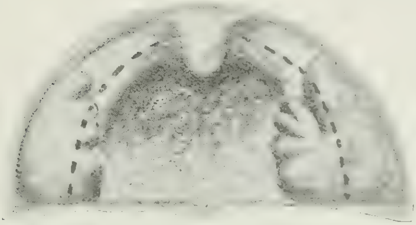
Tonsils enlarged, with infected crypts. History of repeated attacks of tonsillitis. Focal symptoms, pain in extremities. Three X-ray treatments given. After the last there was a return of the focal symptoms; and patient very nervous. Two more treatments given, and symptoms relieved.



b.

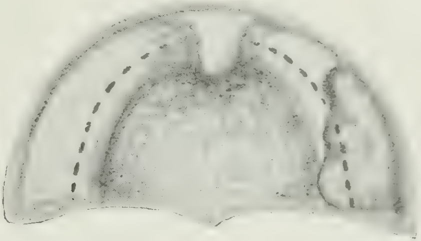
After five X-ray treatments; tonsils apparently two-thirds reduced in size, and retracted behind the pillars. Symptoms relieved. Tonsils not removed in this case.

Fig. 3—Case W. R.



a.

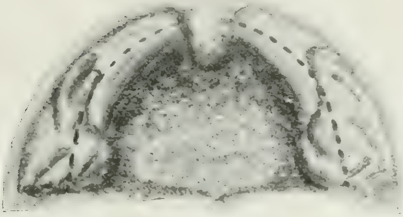
Tonsils enlarged, follicular, with infected crypts. Adenoids. History of repeated attacks of tonsillitis. Sept. 12, 1921, right tonsil and adenoids removed under local anesthesia. Specimen sent to laboratory for examination. Left tonsil given five X-ray treatments.



b.

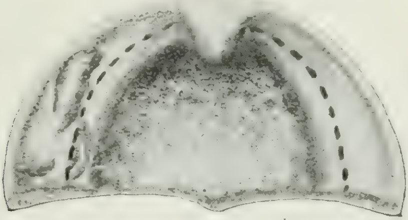
Appearance of throat after five X-ray treatments. Right fossa clear; left tonsil retracted almost completely behind the anterior pillar; with its surface smoothed out; and assumed a normal gray appearance. When this tonsil was removed, it was found to be rather firmly adherent posteriorily.

Fig. 4—Case J. M. R.



a.

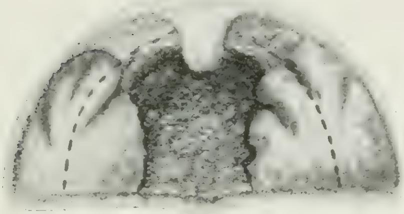
Tonsils enlarged, with infected crypts. Had chronic throat trouble. Sept. 8, 1921, left tonsil and adenoids removed under ether anesthesia. Specimen sent to laboratory for examination.



b.

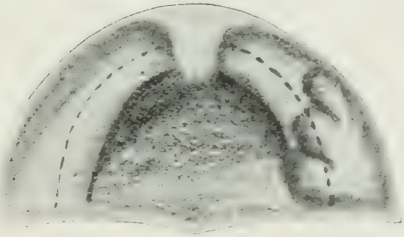
Appearance of throat after five X-ray treatments. Tonsil retracted behind anterior pillar; surface smoothed out, grayish in appearance; crypts open and draining. There was an interval of two months between the third and fourth treatments. Right tonsil when removed was found to be decidedly fibrous in consistency, and densely adherent.

Fig. 5—Case W. H. L.



a.

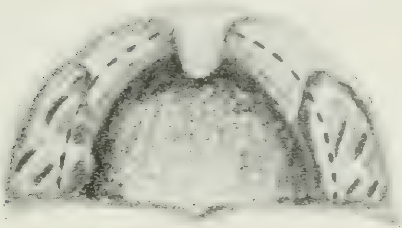
Tonsils markedly enlarged, with infected crypts. History of repeated attacks of tonsillitis. Oct. 11, 1921, right tonsil removed under local anesthesia; and sent to laboratory for examination. Remaining tonsil given five X-ray treatments.



b.

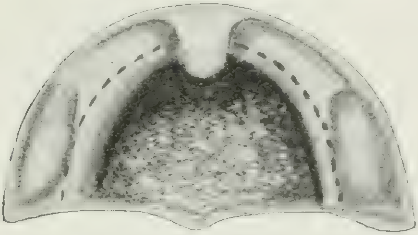
Throat after five treatments. Left tonsil almost one-half reduced in size; this reduction, however, being more apparent than real, as the organ was retracted behind the anterior pillar. Surface was smoothed out, and grayish in appearance. Crypts were open, and even gaping.

Fig. 6—Case J. H.



a.

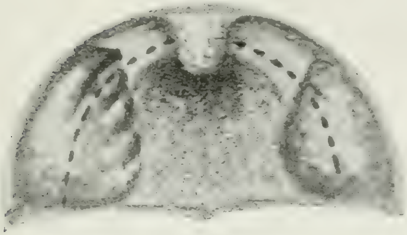
Patient six years of age. History of repeated and severe attacks of tonsillitis. Also had adenoids. Five treatments of X-rays given, one week apart. Nine weeks after the last treatment patient developed a mild case of tonsillitis, lasting overnight. (See text.)



b.

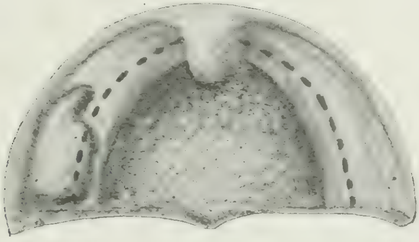
Same throat three months after X-ray treatments. Tonsils retracted behind the anterior pillars. No operative work done in this case; but the change in the general health and the throat condition is similar to those cases in which a tonsillectomy has been done.

Fig. 7—Case J. O.



a.

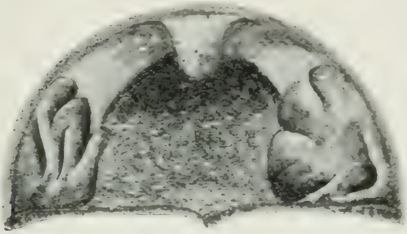
Tonsils very much enlarged; with a large infected crypt in upper pole of right tonsil. History of repeated attacks of tonsillitis and peritonsillitis. Aug. 25, 1921, left tonsil removed under local anesthesia, and sent to laboratory for examination. Five X-ray treatments then given to remaining tonsil.



b.

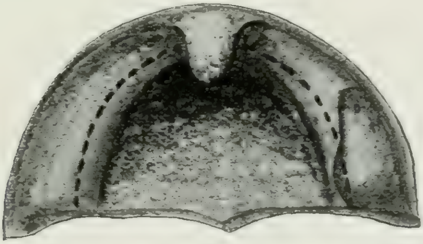
After treatments the right tonsil became more than one-half reduced in size apparently, by retraction behind the anterior pillar. Large crypt in upper pole, gaping and draining. March 2, 1922, this tonsil was removed, and sent to laboratory for comparative study.

Fig. 8—Case H. E. G.



a.

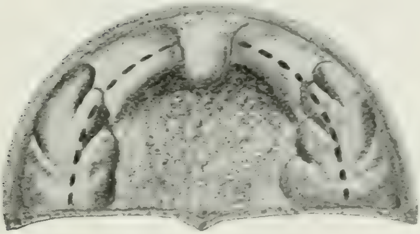
Tonsils markedly enlarged, with infected crypts. Anterior pillars adherent to tonsils. History of repeated attacks of tonsillitis and peritonsillitis. Adenoids. Sept. 26, 1921, right tonsil and adenoids removed under ether anesthesia, and specimen sent to laboratory for examination. Remaining tonsil reveived five X-ray treatments



b.

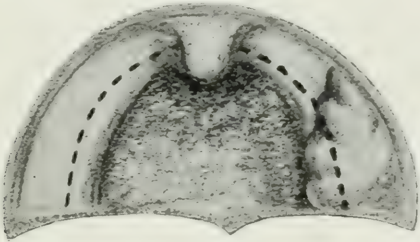
After treatment the left tonsil became apparently more than one-half reduced in size; and retracted behind the anterior pillar. Surface became smooth and gray in appearance. Upon removal on March 29, 1922, the capsule was found to be densely adherent to the constrictor muscle.

Fig. 9—Case N. S.



a.

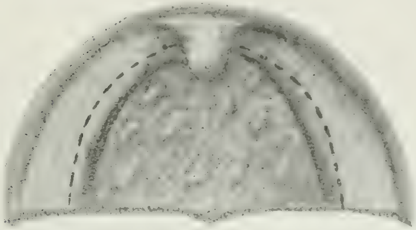
Tonsils markedly enlarged, follicular, with infected crypts. History of repeated attacks of tonsillitis; and a chronic suppurative otitis media, right. Sept. 16, 1921, right tonsil removed under ether anesthesia. Found to be densely adherent posteriorly and above. Left tonsil given free X-ray treatments.



b.

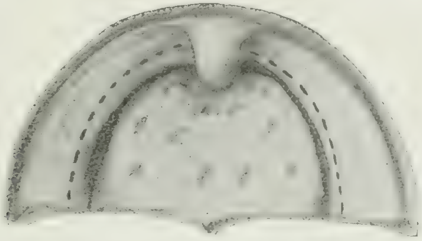
Fifth treatment completed Feb. 10. A month later the left tonsil had begun to show retraction; and the crypts were opened. The otorrhea ceased; and the hearing improved.

Fig. 10—Case M. L. C.



a.

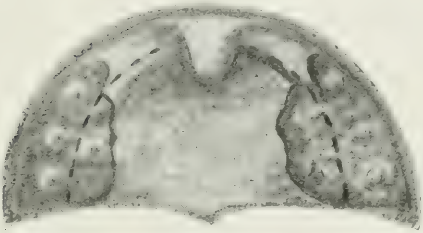
Case of chronic follicular pharyngitis of fifteen years' standing. Tonsils removed in August, 1921, without relief of throat symptoms. Postpharyngeal wall studded with grayish red raised patches; with a distinct red vertical ridge, just behind the posterior tonsillar pillar.



b.

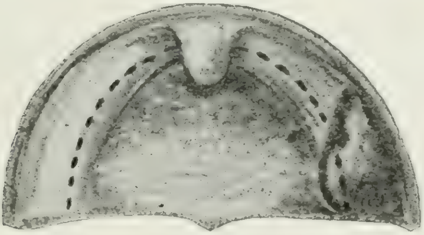
Three months after X-ray treatments, five having been given, the raised patches had become smoothed out, and assumed a dull gray appearance. Symptoms relieved.

Fig. 11—Case H. A. H.



a.

Tonsils very much enlarged; and when soft palate was contracted, the tonsils almost met in the median line. Follicles filled with exudate, and crypts infected. Sept. 29, 1921, right tonsil removed under ether anesthesia, and sent to laboratory for examination. Remaining tonsil given five X-ray treatments.



b.

Three weeks after the last treatment, the left tonsil had begun to show retraction; and in three months' time just a very small portion of the lower half was visible outside of the fossa. The crypts had opened; and the tonsillar surface became smooth and gray in appearance. Remaining tonsil removed March 29, 1922, and sent to laboratory for comparative study.

LXVIII.

OBSERVATIONS UPON THE FORMATION AND FUNCTION OF THE ACCESSORY NASAL SINUSES AND THE MASTOID CELLS.

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The function of the accessory nasal sinuses has been the subject of much investigation and speculation. There is a wealth of literature, illuminating the subject from anatomic, histologic and clinical standpoints, but the fact that modern textbooks still entertain several hypotheses is itself sufficient to betray uncertainty.

The existing theories are here enumerated, not so much for the purpose of pointing out their weaknesses as to emphasize the dissimilarity of any of them to the theory which is the subject of this paper.

Following is a resumé of the several theories which have been advanced from time to time to explain the presence of the sinuses:

I. They impart resonance to the voice.[1]

That they may have some effect upon the timbre of the voice is true, but the ostia are too small to permit of any great influence upon the sound waves. The positions of the sinuses and their ostia are not such as to make them efficient as resonators. Furthermore, large sinuses are by no means invariably accompanied by resonant voices. Guinea pigs and rabbits, for instance, have relatively large sinuses and shrill, irresonant voices.

II. They are remains of certain rudimentary structures which in lower animals are accessory olfactory organs.

Reichwulste[2] believes that the keen sense of smell possessed by certain lower animals is due to olfactory ridges in the frontal, sphenoid and maxillary sinuses.

But Paulson and Hartz[3] find that anosmic animals are often equipped with large sinuses.

Arthur Preyer[4] points out that small children and the lower apes have a very keen sense of smell and no sinuses at all or very small ones.

It may be added that the presence of olfactory nerves or vestiges of olfactory organs has never been satisfactorily demonstrated in the sinuses of man. Jacobson's organ presents such a vestige on the septum, and were the sinuses rudimentary or vestigeal olfactory organs, some similar trace might be expected.

Ingersoll[5] has carefully worked out the anatomy of the olfactory organs of many of the lower animals. His demonstration of the fact that the sinuses or their prototypes in lower animals house certain elaborate organs of smell, does not convince the writer that that function is their essential excuse for existence, nor does it explain why the sinuses persist in man, where no vestiges of these special sense organs are found.

III. They are an adjunct to olfactory function, evenly distributing the inspired air in the olfactory region.[6]

Just how this is brought about is not made clear, but the objections to the previous theory are equally applicable here.

IV. They lighten the bones of the skull in order that proper balance may be obtained.

This theory has been supported by many investigators since Vesalino in 1542 and Highmore in 1681. That they do actually lighten the skull is indisputable, but Braune and Clasen[6] find that, were the sinuses replaced with solid bone, the weight of the head would be increased but 1 per cent. As for balance, our heads are not perfectly balanced, nor do individuals who lack a sinus or two go about with their heads hanging or hold them erect with effort. Furthermore, many quadrupeds whose heads are not even approximately balanced (the horse, the elephant) have large sinuses.

V. They are an adjunct to respiration in warming and moistening inspired air.

In this, however, they are ridiculously inefficient. Even in cases in which the ostia are large and unobstructed, there is no air circulation whatever, and almost no air change. Braune and Clasen have demonstrated that the air pressure varies not more than 8 mm. to 12 mm. between inspiration and expiration. Furthermore, the sinus mucous membrane is practically devoid of glandular tissue and is inefficient in furnishing moisture.

There is, of course, a grain of truth in each of these theories, and the various effects are produced, in a measure, but none of them is in itself sufficient to explain the presence of the rather complicated arrangement and the irregularities in shape and size of the sinuses.

Investigators are satisfied to assume that the sinuses have a function; upon this and little else they agree. If we presuppose nothing, but observe the occurrence of these cavities, the manner, time, position and extent of their incidence and growth in relation to the growth of the individual, certain outstanding facts are encountered.

The first and most obvious of these facts is that the cranium early attains its full, or nearly full, growth, while the face develops slowly and undergoes various changes in shape and in position relative to the cranium. The jaw grows as the deciduous teeth are shed and the permanent teeth erupted. The growing individual has need of a constantly increasing supply of air, the upper respiratory passages widen and expand. The face plays an essential part in these changes.

Froriep[7] measured the cranial and facial portions of the skull and estimated the ratio as

8 : 1 in a term child.
4 : 1 at five years.
2 : 1 in the adult.

The still greater disproportion of cranium to face in the fetus requires only to be mentioned.

The change from the baby face to the adult face consumes roughly twenty years, which coincidently or not, is the age at which the last of the sinuses, the maxillary, attains its full size.

If we examine the mechanics of the situation, the following problem presents itself: Two portions of a bony structure are growing, the one quite slowly, the other quite rapidly. Mechanically we may consider one as standing still, the other growing away from it. For facility in demonstrating this very simple proceeding let us concern ourselves for the moment with the frontal bone.

The frontal bone conforming, dorsally, to the brain, which remains fairly constant in size and position, finds its caudal and ventral portions suddenly called upon to conform to the changing position of the structures beneath it, namely, the nasal and maxillary bones developing in the face.

If the two opposite surfaces of a bone are required to separate, the one from the other, only two mechanical means of accomplishing this are conceivable: (1) The space created by the separation might be filled in with newly formed bone, fat or other substance, rendering it solid; or (2) an air space might be created. This is my conception of what actually occurs. A third mechanical possibility, the creation of a vacuum between the separating surfaces, is not to be considered.

It is not difficult to conceive of this new formed air cavity arising from the nearest existing air cavity, where the line of cleavage begins, and hence lined with similar epithelial covering. This communication is, of course, necessary for the equalization of air pressure, which would otherwise be disturbed by air absorption, by temperature and vascular changes.

In consideration of the rearrangement of the face structures it becomes apparent that the formation of sinuses is necessary for a readjustment to the surrounding parts, and we are not called upon to attribute to these cavities any further functional activity. In other words, the sinus appears not to be pushed out into a bone, bent upon some mission, as is an eye, an ear, or a kidney, but rather that it is "sucked" in from the nearest air cavity by retreating structures, themselves developing to subserve their own functions.

"Both Coffin and Frers[8] refer to the fact that the sinuses are poorly developed in children with adenoids; they consider this due to a lack of normal air pressure in the nose during expiration." I consider it due rather to the underdevelopment of the face—the well recognized "adenoid facies."

This theory, apparently so simply demonstrated in the frontal, is not so obvious in the case of some other sinuses, notably the ethmoidals and the sphenoidals. But even here, if we study the development of the region, and especially the direction of development, these, too, conform to the same mechanics. It is necessary for the sake of clearness to consider the sinuses individually.

The frontal sinus has been already briefly discussed. Major ossific centers appear in the frontal bone at the eighth week in utero. To quote from Schaeffer:[9] "The frontal sinus begins to develop during the fourth or fifth month of fetal life, but it is not topographically frontal until some time after birth. It develops slowly until the seventh or eighth year, then more rapidly up to the twentieth year, and increases somewhat in size up to old age." These periods correspond precisely with the growth of the face. To quote further:[10] "It is frequently stated that the increase of the facial diameters after birth is in a measure due to the pneumatization of the maxillæ, ethmoid, sphenoid and frontal bones by the related paranasal sinuses. While it is true that facial increase is associated with the development of the nasal sinuses, the writer [Schaeffer] is strongly of the opinion that it is erroneous to think of the paranasal sinuses as forcing the face parts to develop or that the development of the sinuses is delayed until all other parts have grown sufficiently to permit of their expansion." He believes "that the developmental processes go hand in hand, and that when a certain time is reached a structure will normally have attained proportions for that period, regardless of related parts."

I agree with Dr. Schaeffer in that it is indeed difficult to suppose that the pneumatization of the sinuses should force the face parts to develop, but the reverse seems quite likely. The face parts develop because the individual has need of them: Larger, stronger jaws, increased breathing space. The sinuses, which, after all, are nothing but unoccupied spaces, result incidentally.

The maxillary sinus begins as a pouch or evagination of the mucous membrane of the floor of the infundibulum ethmoidale, at about the seventieth day of fetal life. From the one hundredth day to term it grows in length from approximately 2 mm. to 8 mm.; in height, from .5 mm. to 5 mm., and in width from a mere slit to 4 mm. In other words, its growth tends toward length and height, not toward width,[11] which follows the development of the face during the same period.

It lies medianward of the orbit and does not occupy the adult position until the eruption of the permanent teeth and the full growth of the upper jaw. It is obvious therefore that when the tooth buds develop into teeth and forsake the maxillary bone for the mouth that a space must be left and that this space is the maxillary sinus.

Note here that the ostium of the maxillary sinus is in the middle meatus,—the point of nearest approach and least resistance to the original position, and not the adult location of the antrum. It was from there that the face-parts first receded and pulled a pocket along after them.

The initial ethmoid evaginations from the mucous membrane of the nasal cavity appear about the fourth month of fetal life. At term true ethmoidal cells exist, but pneumatization of the cells occurs with the growth of the face and the separation of the orbits. Measurements of these capsules from birth to the adult stage, show that their growth, unlike that of any of the other sinuses, is about equal in each of the three dimensions. The frontal sinuses develop upward and outward and forward, along with the advancement of the face; the maxillary grows downward and laterally with the growth of the jaw; the sphenoid not ventrally, away from the nasal cavity, but laterally with the broadening of the adjacent parts.[12] The ethmoids, which lie centrally, follow the development of all these parts and increase in all directions. The question here presents itself: When the face grows away from the cranium and the spinal column, why does the entire nasal cavity not simply enlarge and adapt itself to the new arrangement, instead of sending off the elaborate and troublesome system of accessory sinuses? We have only to recall the picture of atrophic rhinitis to demonstrate mildly the inefficient drainage and humidification accomplished by such a cavity. Furthermore the turbinates which are purposeful organs must be supported in their positions in order to preserve their efficiency.

It is interesting to note in connection with the ethmoidal sinuses that "cells that arise from unlike meatuses never communicate with one another. Furthermore, a cell always communicates with the meatus from which it develops."[13] It is further suggested that the two frontal sinuses never communicate and that two cells of the same frontal sinus never fail to communicate. This is mentioned in passing, merely to strengthen the theory that sinuses are "sucked in" rather than "pushed out" as the rudiments of the purposeful organs seem to be.

The sphenoid sinus in fetal life lies anterior to the sphenoid bone. "No portion of the fetal sphenoidal sinus is contained within the sphenoid bone."[14] When finally it does enter the bone, about the fourth year, rapid growth of the sinus takes place, not dorso-ventrally, as might be expected were the sinus merely pushing its way into the bone, but laterally and cephalocaudally.

In other words the development of this particular region at this time is up and down and crosswise, and not from back to front with the rest of the face. The orbits are separating, the structures of the neck, which are keeping pace with the lateral growth of the jaw and face, are separating the right from the left, and the area of cleavage is that of the sphenoid sinuses.

* * * * * * *

The problem was approached from another angle. If this theory of sinus formation is to hold water, we should find sinuses in all cases in which similar mechanics of development obtain. The skeleton was accordingly searched for bones, opposing tables of which might be required by their process of growth, to alter their relative positions in part, while the remainder of the bone retained its original propornamely, in the mastoid process of the temporal bone.

In the mastoid process cancellated bone is found at birth (as in the sphenoid bone). The socalled pneumatic mastoid is never present at birth, but develops gradually through childhood to adolescence. Here again is a cranium large in proportion to the infant shoulders. Lines of muscle tension are parallel or converge from skull to shoulders. In the adult they are divergent from above downward. A large mastoid process is required to give increased area of attachment and leverage to the muscles. Still the cranial surface of the temporal bone must remain fixed. It is another case of readjustment of opposite sides of a bony structure in relation to one another. Again we have the necessary connection with the nearest air-cavity, the middle ear, and again the lining membrane of that cavity is appropriated.

The work of Wittmaack on the normal and pathologic pneumatization of the mastoid bone[15] throws some light upon the irregularity of sinuses and their formation. Wittmaack contends that the normal mastoid process presents a spongy bone structure until the end of the first year, a partly pneumatized structure until the fifth year and complete pneumatization after that period. All temporal bones which exhibit marked variations from the normal type, show signs of a past pathologic process—an otitis media neonatorum. It is interesting to speculate upon the effect of possible early inflammations upon the sinuses anywhere. Wittmaack states further that inflammatory conditions of the mucous membrane of the middle ear during the first months of life, change the process of pneumatization to one of sclerosis, which corresponds to the first mechanical possibility mentioned in the early part of this paper, namely, that of filling the vacant spaces with bone. Wittmaack finds also that pronounced protrusion of the lateral sinus occurs in complete arrest of pneumatization and only then. Is it not likely, then, that in a bone, the outer table of which is called upon to protrude and the substance of which has by a former pathologic process lost its power of pneumatization—is it not logical that in such a case the inner table should follow the outer, as in this case of the lateral sinus? The protrusion of the lateral sinus is found only in pathologic pneumatization and furthermore the degree of protrusion appears to vary directly with the extent of the inflammatory process.

Several apparently unrelated facts present themselves which have a bearing upon this subject and are worth mentioning here.

There is first the occasional absence of one or both frontal sinuses. In this class are included also those cases in which the sinus is extremely small and confined to its original position in the horizontal naso-orbital region, where to all intent it becomes one of the ethmoidal group. In most, though not all of the radiographs examined, in which the frontals were either absent or unusually small, the superciliary ridge was thick and the individual had "overhanging brows." Measurements of skulls and skiagraphs indicate that in these cases the brow has not grown forward to overhang the face, but rather that the face has, for some cause or other, failed to grow out, and the conditions which create sinuses were not present.

Next to the absence of one or both sinuses we may consider the extreme variations in their size, not only in various individuals, but in the two sides of the same individual. If sinuses were functioning organs, or even vestiges of functioning organs, it should be expected that in company with other paired organs eyes, teeth, kidneys, testicles—they should be of fairly constant size in individuals of the same race, and still more so in both sides of the same individual. If we consider the sinuses as forming, however, as a rift in a log is formed by a driven wedge, it is easy to see how a slight variation in tension produced by the wedge or a slight inequality in the elasticity of the wood, would result in a very large variation in the size of the rift. Thus minor variations in tension, due possibly to differences in the thickness of the bone, density, degree of ossification, or what not, would account for the wide variation in the size of the resulting cavities.

Skulls are encountered, in which the sphenoidal sinus of one side is rudimentary, and the sinus of the other side occupies the entire body of the sphenoid, necessarily crossing the middle line to do so.[16] This alone suggests that the mechanical requirements of the situation demand a pneumatization of the bone and that upon the failure of one sinus to develop (through any cause, say an early inflammatory condition, such as Wittmaack describes in the temporal bone), the other sinus is drawn in to fill the gap. Such variability and adaptability is characteristic of the structural elements of the body rather than of functioning organs.

Along with the absence and variation in size of the sinuses may be considered the enormous frontal sinus of the elephant, which occupies not only the usual position, but traverses the entire cranium in the midline. Here is a striking example of unusual facial development required by the muscle attachment of the trunk and by the tusks, tending away from a practically stationary cranial cavity.

Nowhere else in the skeleton do we find the requirements of disproportionate growth after ossification that exists in the skull and are met by the formation of sinuses.

To summarize: 1. The nasal accessory sinuses and the mastoid antrum are apparently the result of a plastic rearrangement of the skull necessitated by the disproportionate growth of the face and cranium and associated structures after they are fully or partly ossified.

2. They are cavities left by the separation of two walls of a bone.

3. They must communicate with the external air to avoid physical disturbances; they do this by communication with the nearest existing air cavity (from which they arise); they are lined for this reason with the membrane of the cavity from which they arise.

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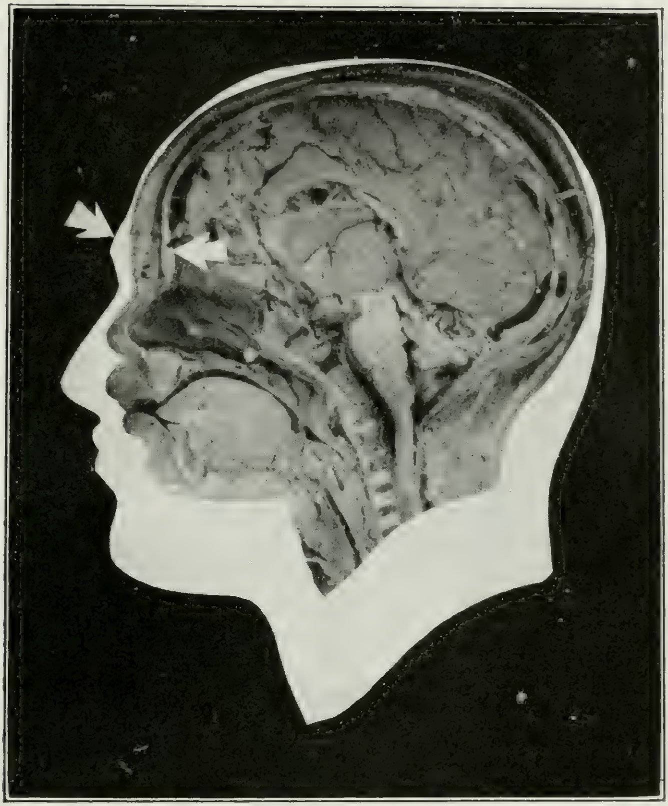

Fig. 1.

Outlines of cranium and face of a newborn, photographed to scale and superposed upon the outlines of an adult head in such a manner that the crania practically coincide. Note the difference in angle and size of face as compared to cranium. The arrow on the right indicates the surface of the frontal which is required to remain fixed. The arrow on the left indicates the position which the outer table of the frontal bone is called upon to assume in the adult.

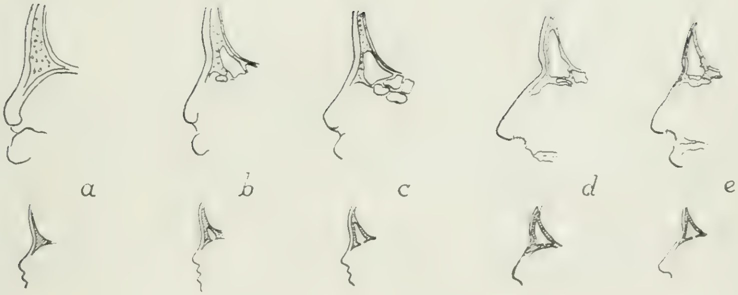

Fig. 2.

Outline drawings indicating the development of the frontal sinus at various ages. The diagrams beneath indicate the separation of the two tables of bone in the process, a, newborn; b, 2 years; c, 7 years; d and e, adult types.

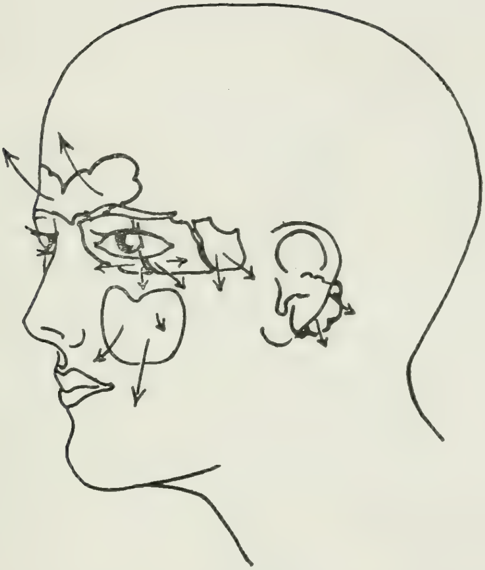

Fig. 3.

Diagrammatic representation of the directions of greatest development of the sinuses, as detailed in the text.

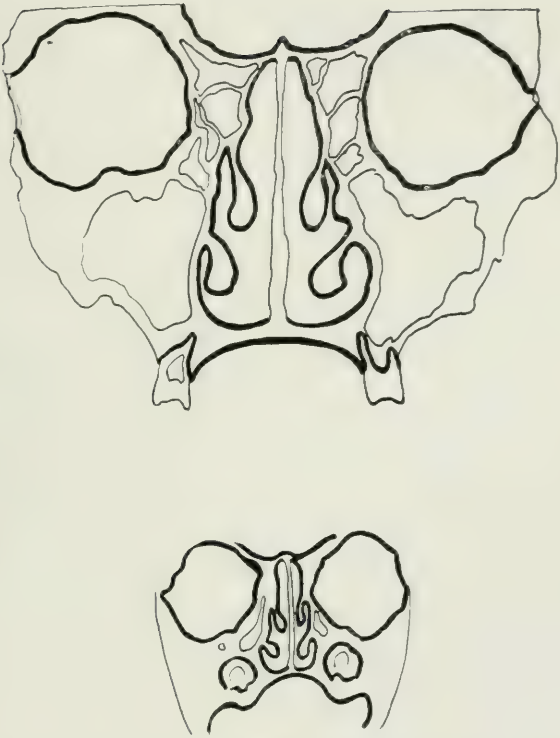

Fig. 4.

Outlines of the cavities containing purposeful organs are accentuated: the brain cavity, the eye cavities, the nose cavity, the mouth cavity. The upper figure presents the adult relations; the lower, those of the infant of one year. It will be seen that the various organs, closely related in infancy, but receding from one another as the fact grows, leave spaces between them—the accessory nasal sinuses.

LXIX.

OPERATION OF TOTAL LARYNGECTOMY FOR THE CURE OF INTRINSIC CANCER OF THE LARYNX.

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I am convinced from personal experience that the operation of total laryngectomy for the cure of intrinsic cancer of the larynx should take a foremost place in the surgery of cancer in general. In no other location in the body have the results excelled and in few have they even approached these in the field under discussion.

From this assertion the surface cancers might be excepted, since their less difficult and earlier diagnosis give surgery and radium an opportunity not secured to cancer in other and deeper parts. Though, technically speaking, cancer in the larynx may be classified as a surface disease, clinically, on account of its hidden position and high malignancy, it deserves a place with the socalled deep seated cancers.

The failure in the past to cope with this horrible death—and I can conceive of no ending more disgusting or more painful—was due to the inexperience of the general profession. An operative mortality of over 30 per cent and the high percentage of recurrence did not encourage either the surgeon or patient to face the ordeal. A high degree of supineness fell upon the profession, which expresses itself in pessimistic utterances often, I regret to say, from those who should have been foremost in leading the fight. The loss of voice incident to the removal of the larynx was a stumbling block to both doctor and patient. They seemed to ignore the fact that the loss of voice was inevitable, in the progress of the disease, coming long before its fatal termination.

Coupled with this was an almost total ignorance on the part of the public on the one very simple manifestation of laryngeal cancer—i. e., hoarseness or change in the quality and character of the voice in the middle years of life. If this one simple manifestation could be branded on the consciousness of the practitioner and of the public alike, thousands of cancer victims could be saved yearly.

If we except the rare fulminating malignancies in which all effort is generally futile, intrinsic cancer of the larynx is, in its beginnings and often in its more developed stage, still a local diesase. It is cut off to a considerable degree from the lymph channels of the neck by a cartilaginous wall which acts for a time as a barrier to extension. Two vulnerable directions of extension exist: One backwards towards the esophagus and one upwards towards the epiglottis and lateral hypopharynx. Rarely in the early stage does the disease extend directly through the cartilage to the tissue of the neck.

After many years of observation and operative effort in this field, one can with fair precision prognosticate the ultimate result in any given case.

The factors tending towards an optimistic outlook may be set down as follows:

1. Slowness of growth.
2. Freedom from involvement of the posterior part of the larynx.
3. Superficial growth. Cancer starting in the deeper layers of the larynx may not be more malignant but they often escape detection until well developed.
4. Extension forward and downward rather than upward and backward.
5. Age of patient. Cancer in the late thirties or early forties is more malignant than in later life.

Arytenoid involvement, in my opinion, places the disease on the borderline of the extrinsic class and tremendously lessens the hope of cure. Biopsy for diagnosis is to be avoided if possible. It has been resorted to only a few times by me in eighteen years, principally where syphilis masked an underlying cancer. In these luetic cases the suspicion of malignancy was confirmed by biopsy.

The diagnosis should be made on the history, appearance, behavior of the growth and on the exclusion of syphilis and pulmonary tuberculosis. It may seem trite to state that the loss of motility in the affected cord is almost a pathognomonic symptom of cancer. This is due to fixation of the musculature by infiltration. The disease attacks one or the other cord, generally in its middle third. I have seen no primary involvement of the interarytenoid space so characteristic of tuberculosis. The Leitz arc lamp is of great assistance in securing a clear definition of the disease.

The extent of the growth must not be estimated by the image seen in the laryngeal mirror or even by the more accurate impression secured by direct laryngoscopy. The upper edges of the growth are all that appear for inspection, the extension downwards and inwards being hidden from view. Growths of deep origin are the most deceptive. I believe that it would be quite safe to add two-thirds to the visible growth in forming a mental picture of the size.

Formerly when in doubt on the extent of the disease, I advocated opening the larynx for better orientation. I am now opposed to this procedure and believe that it should be avoided, since the incision may bisect the growth and disseminate it. Furthermore, this procedure may and often does let blood into the trachea and adds just that much more to an already serious and dangerous operation.

If doubt exists in the operator's mind as to the extent of the growth, the patient should be given the benefit of that doubt in having the more radical operation done.

It is my firm belief, founded on experience, that only the most incipient cancers should be treated by any method other than the most radical.

Statistics will bear me out in asserting that secondary operations for cancer are almost futile in any field. You have but one shot for a bull's eye and, if you miss, the patient pays with his life.

Therefore, I assert, if we are to get anywhere with this terrible disease we must divorce our minds from a lot of sentimentalism born mostly of inexperience relative to the socalled pitiable plight of the laryngectomized patient. I have been in intimate contact with these socalled victims for years, have seen them in their business and social lives and have observed nothing but cheerfulness and gratitude on their part at their deliverance from certain death. My experience is leading me yearly further away from compromise in dealing with intrinsic cancer of the larynx. It has also taught me to leave the frankly extrinsic cases to their fate. For five years I have so stood. The extrinsic cases done prior to that time have all since died of cancer, so have many of the window resections and hemilaryngectomies.

Here I might digress and say a word on some amazing statistics of cures by thyrotomy, and even by excision under suspension. Is it possible that the general profession in the regions from which these statistics emanate is more enlightened and more alert in the detection of laryngeal cancer than we are in New York and its environs? To believe this would surely be unpatriotic, even if it were not very improbable. Why do we in New York so rarely encounter the incipient case, seemingly so common elsewhere? If total laryngectomy on early intrinsic cases has cured in my hands, say 66 per cent, why does thyrotomy and suspension excision yield in other hands even a larger percentage? If true, then the part must be greater than the whole.

The question must arise in the unbiased mind: Are we laryngologists facing the surgical facts in dealing with laryngeal cancer, or are we compromising with the patient's fears and desires?

Surgery in other fields for cancer strikes wide of the disease. Do you believe that the general surgeon would be content with a quarter of an inch leeway on a malignant growth or feel that he had accomplished his duty in so operating? Those who advocate thyrotomy and suspension removal for the majority of laryngeal cancer seen by them must be operating with even less than a quarter of an inch leeway.

We laryngologists have much to atone for in our past. Witness the long and fruitless discussion on the relative merits of tonsillectomy versus tonsillotomy. Think of the years it has taken to place sinus disease where it now stands. Do you realize that thousands of submucous resections and partial or total turbinectomies are even now being done for nasal obstructions due solely to sinus disease and tonsillar infections? It looks as though we are learning little from the past and are still stuck in the mire of tradition on this question of the best method of dealing with laryngeal cancer. My personal position on this question is that thyrotomy is rarely indicated, that radium is on trial and that total laryngectomy is the logical, safe and tried procedure of choice.

STATISTICS.

Six hemilaryngectomies were done prior to 1916-17. Two are now living. Four died of recurrence within two or three years of the onset of the disease. The two surviving cases were operated upon at a very early stage. I now know that the others should have had a total laryngectomy, as they were not incipient cases as I now understand the term.

Thirteen total laryngectomies were done prior to 1916-17. Two of these were diabetics and succumbed to the operation. Six were advanced intrinsic cases with involvement of one or both arytenoids. Four of the six died of recurrence. Two are still living. Three were intrinsic cases (arytenoids free). One of these cases died from a recurrence, one from nephritis one year after operation and one is still living.

The extrinsic cases with involvement of the arytenoids, the anterior lip of the mouth of the esophagus or the glands of the neck, have all died of recurrence.

Twenty thyrotomies were done between 1906 and 1920. Eight of these had the growth removed, leaving the cartilaginous wall. Seven died of recurrence, one is still living. In 1911 the window resection was taken up. Twelve were operated on by this method. Eight of these had recurrences and died. A subsequent total laryngectomy failed to save three of the eight. Four are still living, two after three and two after two and a half years.

Looking back upon this series, I am convinced that but few of these twenty cases were subjects for anything short of total laryngectomy. With my present experience, several might have been saved by the more radical operation, since many of them were well beyond the incipient stage, yet intrinsic.

Since 1916-17 only two thyrotomies have been done, both on extremely early cases. As stated above, they are still living, without recurrence after two and a half and three years.

Since 1916-17 I have seen 143 cases of laryngeal cancer. Forty-six of these were still intrinsic and gave some hope of permanent cure. The disease was confined to the laryngeal box. The arytenoids were partially involved or not involved at all. Three cases were truly incipient and might have been suitable for thyrotomy. In the 97 remaining cases the disease had extended beyond the larynx in various directions. In many the glands of the neck were palpable.

Thirty-one out of the 46 intrinsic cases came to operation for total laryngectomy and two for thyrotomy. In these 33 cases there were no surgical mortalities. There have been no operative deaths since the two reported five years ago. Twenty-nine were done by the one stage operation; two by the two stage operation. Twenty-five have entirely discarded the tracheal canula, an advantage rarely experienced in the two stage operation.

One case of sarcoma appears in this series. The oldest case was 80, the youngest 41. Two were females. Primary union was secured in only five. A large defect occurred in two, requiring a subsequent plastic. Since three years no plastics have been necessary, owing, I believe, to improvements in the operative technic and to better management of the postoperative infection.

The series reported in 1916-17 were mixed cases, some advanced, some early. The 33 since that date are all fairly early cases. All extrinsic cases were rejected, for the good reason that in ten years of surgical effort, from 1906 to 1916, I had not succeeded in ultimately saving one. I am convinced that extrinsic cancer of the larynx cannot and never will be curable by surgery.

Twenty-six of the thirty-three cases are still alive, with no evidence of recurrence. One died at 84 years of age of pneumonia. One died two years after the operation of cerebral hemorrhage.

One of the most hopeful died of recurrence three years after the operation, and four cases in which the disease was well back towards or partially involving the arytenoids have succumbed to recurrence within two and a half to three years from the time of operation. Fourteen cases out of eighteen show no recurrence after a period of from three to five years.

If five more cases in this series of 33 should succumb to cancer, making in all ten cancer deaths in 33, we would still have 66 per cent of cures, a showing quite worth the effort.

TECHNIC.

Since my last publication on this subject, over five years ago, the operative technic has been improved in several points though the rationale of the operation is essentially the same. The troublesome plastic repair for closure of the hypopharyngeal defects in the earlier cases have been almost entirely eliminated in the latter ones. Convalescence has been materially shortened. Though some infection in the wound is the rule I do not now so often see the extensive gangrenous sloughing which made a few of the earlier cases so difficult. In 31 consecutive total laryngectomies and two thyrotomies neither mortality nor serious complications have occurred. The shortest wound healing was ten days and the longest five weeks. Twenty-nine were done by the one stage operation. Two required a preliminary tracheotomy. Twenty-five have been able to dispense with the tracheal canula. In three it must be retained, since the tracheal opening collapses when the tube is removed. Three could dispense with the canula but fear to do so.

The two stage cases cannot dispense with the canula, as the tracheal rings at the opening were destroyed by the tracheotomy. To secure a tracheal and skin union with a tracheal ring immediately beneath is one of the aims of the one stage operation. This gives a rigid opening, dispenses with the canula and adds immensely to the patient's future comfort. After no two stage operation have I seen this result secured.

I have been much gratified with the speech results. All but one of the cases have developed a whisper audible at several feet in a still room. One operated seven years ago has acquired loud speech and can talk over the telephone. He can count up to twenty on one stomach full of air. In some way, known only to himself, he opens his esophagus, fills his stomach with air and makes audible speech by expelling the air slowly. Several are engaged in large enterprises, which they conduct as successfully as they did before their operation. Some have informed me that their stenographers take their dictation with ease. They do not complain of discomfort during the cold months, not even in Canada. To protect the lungs against cold dry air, a bulging wire screen covers the tracheal opening and extends several inches down and across the chest. Above this a high soft collar is worn to exclude the external air. The patient breathes the warm moist air coming from beneath the clothing. This simple device has done much to protect the trachea and lungs during the cold months.

The surgical period may be divided into three stages:

1. The preparation.
2. The operation and its technic.
3. The after-treatment.

I.—PREPARATION.

Two deaths have occurred in seventy operations. These were among the earlier cases before a rigid examination and preparation were instituted. These deaths were due to sugar retention. One was a frank diabetic and was treated by the more or less crude method then at our disposal.

The false assumption was that a sugar free urine put the patient in a safer operative state. He died of an ischiorectal infection (three weeks after operation), secondary to a general septicemia. The other death occurred in an individual with a normal urine. The history pointed to a glycosuria occurring two years previous to the time of operation. The condition following operation pointed to diabetes. A blood chemical revealed retention with a high sugar index. He died of a septic bronchopneumonia.

The lesson learned from these deaths has since saved several cases from a similar fate. This leads up to the first point in the period of preparation. The patient is put in the hands of a metabolist, who sees that he is chemically balanced. If retention exists, especially of sugar, treatment is instituted. If sugar cannot be brought to a safe level the case is rejected. Several cases have thus been brought through which under less careful treatment might have succumbed. It is gratifying to observe that this procedure is now extending to all branches of surgery.

Where the heart is weak, digitalization is a very essential part of the preparation. Special attention is paid to the colon. For one week preceding the operation, every other day a weak carbonate of soda enema is given, followed by a saline colon irrigation. A low protein diet, excluding eggs, beef and sweet milk, is essential in all cases for a week or two preceding the operation.

A competent dentist extracts all carious teeth and pyorrheal teeth and cleans those remaining. So essential is this that it should be entrusted to only the most expert and conscientious. Where all teeth are absent a primary union in the wound may be expected. The neck infections from a filthy mouth may take on the picture (so seldom now seen in surgery) of hospital gangrene. It occurred in a few of the earlier cases. Healing after such an infection leaves a tremendous gap in the throat and has in several cases required an extensive plastic for repair.

Before undertaking a plastic repair, the wound must be entirely clean and cicatrized. Impatience to secure a quick closure dooms the whole procedure to failure. It is also essential to use local anesthesia, since the vomiting incident to a general anesthesia is very liable to cause infection.

Adhering to these principles, I have succeeded in closing the defect in all cases in one operation. The feeding tubes must be used as described in the first operation. If sinus disease exists every effort should be made to make the nasal cavities as clean as possible, not by operative methods, since this but stirs up a latent infection, but by suction and suction douche twice daily during the period of preparation. It is some consolation to know that many of these old sinus infections are not very virulent.

It is my custom to give bicarbonate of soda for a few days preceding the operation.

2.—THE OPERATION AND ITS TECHNIC.

The surgical principles to be kept in mind may be taken up under the following headings:

1. Anesthesia.
2. The prevention of the inhalation of blood.
3. Cleansing and disinfection of the mouth, pharynx, nose, hypopharynx and the prevention of their secretions from contaminating the wound.
4. The secure anchoring of the tracheal stump.
5. Drainage.
6. Method of closing the wound.

7. Method of excluding the wound from the tracheal secretions.

8. The proper placing and securing of the feeding tube.

1.—ANESTHESIA.

A combination of local and general anesthesia is in my opinion better than one of these alone.

By this method the duration of the general anesthesia is reduced to one-half hour, an important factor in the resistance of the patient.

Where the growth encroaches upon the breathway, the giving of the general anesthesia from the start may increase the embarrassment and force a tracheal opening before the surgeon is ready. If the patient becomes cyanosed and is not promptly relieved, the consequent lung hyperemia may predispose him to a postoperative pneumonia. Hence the advantage of skeletonizing the larynx and upper two or three tracheal rings under local anesthesia before the general anesthesia is given. One per cent novocaine is used for the skin and one-fourth of 1 per cent for the deeper structures. To this is added a very minute amount of adrenalin.

The T incision is used. It has many advantages over the linear incision. In fat necks it is indispensable. It lends itself better to drainage. The angles where the lines cross can always be made to heal by primary intention, thus preventing postoperative fistula by closing the upper central portion of the wound.

The dissection is carried backward until the larynx and trachea are skeletonized. When hemostasis is complete and all vessels tied the patient is given a general anesthetic. The trachea is now cut across just below the cricoid, taking minute care that no blood enters the lumen of the tube. It is an advantage to inject between two rings into the trachea before dividing it, a few drops of 10 per cent cocain solution. This prevents coughing. The larynx is lifted forward and the posterior wall of the trachea is incised down to the esophageal wall. A rubber tube which fits snugly into the tracheal lumen is inserted into the trachea to a distance of about two inches. This acts as a tracheal extension, turns back the blood and enables the anesthetist to continue his anesthesia without being in the way.

The larynx is separated from the esophagus from below upwards to a point behind the arytenoids. Then it is allowed to fall back into position, the thyrohyoid membrane is divided, opening into the hypopharynx just below the attachment of the epiglottis. Before this opening is made, the anesthetist or an assistant opens the mouth wide, sucks out all secretion and paints the entire cavity, the pharynx and the hypopharynx, with a 2 per cent solution of mercurochrome. Iodin on mucous membrane causes desquamation and predisposes to infections.

The nasal cavity is similarly treated. The edges of the opening through the thyrohyoid membrane are grasped and held apart. A yard of gauze folded two inches wide is stuffed into the hypopharynx and crowded upwards until it fills the mouth and pharynx. At this point a careful inspection is made of the growth. If it is found to be entirely intrinsic the larynx is removed by cutting as close as possible to the superior border of the thyroid cartilage. The opening thus made in the hypopharynx is small and better lends itself to successful repair. If the disease has approached the top of the laryngeal box or has involved the arytenoid, then more tissue is sacrificed, even to the removal of the anterior esophageal wall adherent to the posterior surface of the larynx. In several cases one to two inches of this anterior part of the esophageal lumen has been taken away with the larynx without producing subsequent stricture.

Just before the last stitch is tied in the closure of the hypopharynx the anesthetist removes, through the mouth, the gauze packing filling the mouth and the pharynx. The mouth and pharynx are again cleansed by suction and again painted with a solution of mercurochrome (2 per cent) A feeding tube of a size which will pass through the nose without undue pressure is passed through the more open side of the nose. When its point appears in the esophagus beneath the untied stitch the surgeon directs it into the esophagus to a depth of six inches. The point of exit from the nose is now carefully marked and the tube secured to the face by adhesive plaster.

The last stitch is now tied. If the redundancy of the tissues permits, a second layer of stitches is placed over the first in the hypopharyngeal closure. No. 1 plain gut is used.

The trachea is anchored to the skin of the neck by two or three stay sutures, each passed around a ring and brought out about one inch from the edge of the wound. These are tied on small perforated lead discs. This steadies the tracheal stump in the wound and relieves the strain upon the stitches which are to unite the skin edges with the mucous membrane of the trachea. To make this union more exact the fat along the skin wound edges on both sides is cut away. This allows the skin to fall more easily into relation with the edge of the tracheal stump. The skin strip and edge of the trachea are united by interrupted stitches, using fine silk, or better, fine equisetene. The wound is loosely closed, no effort being made to bring the deeper parts into the anatomic order. It is assential to get primary union at one point—that is, where the two lines of the T cross. I have observed that if the integrity of this part of the wound can be maintained the subsequent healing is much more rapid and hypopharyngeal fistula does not occur. If this point breaks away or, for the securing of better drainage, has to be opened, an effort should be made as easly as seems prudent, to again bring these angles of the T into place.

Silver wire here serves better than any other suture material since it may be left in situ for a long time, even in the presence of infection.

A tube and gauze drain are passed into the depth of the wound at the ends of the bar of the T. Just above the point where the trachea is secured to the skin two small gauze drains are placed, one on either side. This guards the direct route of the medistinum.

A large tracheal canula (36) is wound with gauze impregnated with bismuth paste. The winding is so fashioned as to form a conical cork. This is inserted into the trachea and should fit as a cork does a bottle. The object of this corking the trachea is to prevent tracheal secretions from contaminating the wound from below and to turn back from the trachea any wound secretions from above. It also protects the tracheo-skin union. It has been in my hands a very serviceable device, especially later if infection arises and wound drainage becomes profuse. Without this tracheal plugging lung infection would be almost inevitable.

The wound is dressed in the usual way. A rubber apron is placed over the end of the canula and catches all tracheal secretions.

During the repair period of the operation the patient is given little or no anesthetic. Deep anesthesia is imperative only from the time the trachea is opened until the hypopharynx and esophagus are closed.

AFTER-TREATMENT.

The immediate treatment usual after any major operation is carried out. I will speak only of the conditions peculiar to this operation. It is here that the skill and experience of the surgeon are often taxed to and even beyond their limit. The after-treatment in laryngectomy cannot be delegated to an assistant or a member of the house staff. Painstaking, constant care on the part of the surgeon is the only key to success. If infection occurs the surgeon must be at least one jump ahead of it. The reverse means almost certain death to the patient. I attribute the prohibitive surgical mortality of a few years ago in laryngectomy to four causes, viz., a careless preparation of case, prolonged general anesthesia, carelessness in the prevention of blood entering the lungs during the operation and mismanagement of the septic infection so common after operation. Another factor may be added. Rectal feeding and drop feeding by mouth were depended upon prior to my demonstration that the esophagus would tolerate a permanent tube for weeks. Rectal feeding was one of the greatest fallacies that ever became rooted in the professional mind. It died a very long, hard death and I, for one, sincerely hope that it is dead indeed and forever.

The local reaction in the neck is considerable and may simulate infection. If doubt exists in the surgeon's mind he would do wisely to extend as widely as possible his drainage openings and keep a wary eye out for extension along the muscular planes of the neck. If this occurs it must be followed and opened widely.

Later if pus becomes profuse and if a break takes place in the esophagus, suction to remove these secretions is of inestimable value. Tubes are placed at the bottoms of the pockets and through the opening in the esophagus. These project through the dresssing and the nurse applies suction to these tubes as frequently as is indicated. Dakin's solution has been useless in these cases since it may get into the trachea and cause intense irritation. I prefer a 2 per cent mercurochrome solution used sparingly.

If, as sometimes happens, trachitis follows the operation, suction applied through a catheter unloads the trachea and prevents gravitation pneumonia. With the larynx gone normal expulsive cough is obliterated, hence the impossibility on the part of the patient to unload his trachea and bronchi. Here suction carefully and properly applied has, in my hands, saved many lives, not only in this operation but in all conditions where the trachea is open and the bronchial tree loaded with secretion.

FEEDING.

Much of our success in piloting these cases across the postoperative period is due to the permanent feeding tube. Feeding to the point of toleration discourages septic infection. Of what avail are the feeble efforts of the surgeon against infection in a starving patient? For over twenty years I have used this method of feeding without one untoward result traceable to its use.

At first, excepting esophagitis, I used it gingerly and for short periods. I could get no information on the subject excepting that all seemed afraid of it. In Vienna, in 1911, it was not used and the men in this work advised against it. The drop method of feeding was employed. They would not discuss its merits with me or be influenced by the years of my practical experience, as I was an American.

Feeding is begun just as soon as the anesthetic period is over. For two days from one-half to two-thirds the caloric requirements are given, then the amount is increased to tolerance.

Any well balanced diet capable of being reduced to a fluid or semisolid may be used. I append two diet lists founded on the caloric requirements of a normal individual of average weight. Fruit and vegetable juices are essential and must not be kept out of the dietary longer than five or six days.

Gravitation serves for ordinary liquids. For thicker ones the piston syringe may be used. The patient being propped up in bed, the food is very slowly introduced. After each feeding (which may be once in two and a half to four hours) a few ounces of water are passed through the tube to cleanse it. Several times a day the pharynx is cleansed of mucus by suction and the mouth and teeth cleansed with mouth wash and brush. Dressings are changed as often as need be to keep the wound free from secretions. In foul cases the wound packing may require several changes in twenty-four hours. The patient must be shifted from side to side and encouraged to sit propped up in bed.

COMPLICATIONS.

1. Pneumonia.—The entrance of blood into the lungs during operation and prolonged deep anesthesia are the two most noteworthy causes of pneumonia. One case of bronchopneumonia occurred in a series. It was in a diabetic and proved fatal.

2. Mediastinitis.—This is said to be one of the dangers of this operation. It has not appeared in my cases. Liberal drainage at the time of the operation and free opening of the wound at the onset of injection must materially minimize this danger.

3. Extensive gangrenous sloughing of the wound may produce great debility, but has not in my experience caused death. On the other hand, it has perhaps saved life by eradicating more efficiently than the knife any wandering cancer elements in the neck.

4. Hiccough.—A troublesome hiccough has occurred in four of my cases following total laryngectomy. In only one did it develop a serious aspect. In this case small doses of apamorphin controlled it after all other means failed. It is probably due to irritation of the vagus nerve in the operated field.

5. Hemorrhages.—Secondary hemorrhage immediately following operation is due to a defective technic on the part of the surgeon. I have made it a rule that all clamped points in operation in the neck must be tied. If a good pedicle cannot be secured for tying then a transfixion stitch is used. Second-

ary hemorrhage from sloughing rarely occurs from a large vessel since the walls of the larger vessels are very resistant to infection. It is easily controlled and not serious unless prolonged. There is a type of secondary hemorrhage in healing wounds just past the septic stages which is extremely difficult to control. Septic absorption so changes the chemistry of the blood as to convert the patient into a temporary bleeder. The blood becomes thin and clots poorly. In this state of the patient the granulating surface of the wound may bleed profusely. The granulations at the point of bleeding should be curretted away and the patient transfused. Perhaps repeated small transfusions act better than one large one. I have witnessed this complication in one case recently. It did not yield to transfusion. The bleeding lasted for two weeks, the hemorrhages occurring about three times in twenty-four hours. It was finally controlled by the complete removal of the over active granulation tissue in the wound.

General Sepsis.—One case in my series died of general sepsis. He was a diabetic. He died three weeks after the operation from an ischiorectal abscess and gangrene of the peroneum.

Tracheitis.—A mild degree of tracheitis is frequently observed after laryngectomy. Rarely this may be severe enough to threaten the lungs and produce a secondary bronchopneumonia. It is here that suction to clear the trachea and upper bronchial tree from secretion is of invaluable aid.

The fight to place laryngectomy on a firm basis, both from a surgical and a life saving standpoint, has been an uphill struggle. We have been hampered by an apathy on the part of the profession engendered by the prohibitive surgical mortality in the past and an almost hopelessly large percentage of recurrences.

Prominent men in our midst have decried the operation since, failing themselves in its successful performance, they could not conceive of its success in other hands. Hopeless cases, often in the septic stage of the disease, were operated upon and forced to pass through two deaths instead of only one had they been left alone.

Radium in the hands of the unscrupulous and the unscientific has beguiled the poor victims to their doom and deprived them of their only chance, an early and radical operation. Scientific observers like Dr. Quick have made no extravagant claims for radium in this disease.

Partial removal of the larynx or attempts to remove the growth by thyrotomy or suspension in cases entirely unsuited to these procedures, have heaped discredit upon the whole procedure.

Finally, there has been the failure of the profession at large to realize that prolonged hoarseness in a person of cancer age may be, and very often is, the first sign of danger, demanding immediate intelligent and painstaking investigation by one competent to differentiate between the simple and malignant disease in this field. If cancer has its inception external to the laryngeal box its early detection must be, till more light on this disease is vouchsafed us, of little avail to the victim. In my opinion, we are helpless in this solution. Hence the only cases where early detection is of importance are those (the intrinsic) in which hoarseness may be an early symptom.

Permit me in closing to reiterate two statements. Incipient intrinsic cancer of the larynx is a very hopeful disease if we apply to its cure the same horse sense exhibited by the general surgeon in the treatment of cancer elsewhere in the body. The general surgeon operates radically. The laryngectomized patient is not, as many state, only a surgical curiosity, a derelict, a miserable thing apart from all his kind. He is usually a useful, active citizen, capable of continuing his life's work, of supporting his family and realizing, if not to its full extent, at least to a large extent, the joy of living.

LXX.

ABSCESSES OF THE LARYNX AND TRACHEA FOLLOWING INFLUENZA.

By Clement F. Theisen, M. D.,

Albany.

No attempt was made by the writer to cover the literature on this subject. It is an almost impossible task, and it is questionable whether the report of many cases, and I do not know whether there have been many cases of this kind, is of any great interest, so that only a brief review of the writer's own cases will be given in this paper.

Judging by the fact that the writer has been able to find records of only six cases of abscess of the larynx and trachea, occurring during epidemics of grip and influenza, in his own practice, during the past twenty-five years, this complication must be fairly rare.

From an etiologic standpoint these cases are of particular interest, mainly because they are so difficult to explain. Four of the cases came under the writer's observation during the severe influenza epidemic that swept the country during the winter of 1917-18. None of us will forget the appalling death rate of that epidemic very soon.

One case was seen during an epidemic of influenza twenty-four years ago and one during the epidemic of the past winter which perhaps might be properly called "grip." It was certainly nothing like the influenza epidemic of 1917-18, at least not in Albany. But while the mortality rate was not nearly so great, I think we will all agree that there were almost as many ear and sinus complications as in the winter of 1918, but, in the writer's practice, not as many serious throat complications as in 1918. The writer's six cases were all adult males between the ages of thirty and forty-two years.

The bacteriology of five of the cases was worked out. In none of the cases was the influenza bacillus found. In three, cultures showed mixed staphylococcus and streptococcus aureus infection, and in the other two streptococcus aureus.

I suppose these abscesses in the larynx and trachea following influenza are mainly the result of the run down condition of the patient. We could understand the etiology better if the larynx and trachea contained some of the same kind of lymphoid tissue that there is in the pharynx, which is so subject to infections of all kinds. There is of course very little lymphoid tissue in the larynx and trachea. I suppose these abscesses are in a way analogous to the abscesses of the lateral columns of the pharynx. We have all seen many times, particularly during grip and influenza epidemics, abscesses in apparently clean throats. I mean in throats from which all the lymphoid tissue, tonsils and adenoids, had been thoroughly removed. It is impossible to remove all the lymphoid tissue from the pharynx, and in throats from which the tonsils and adenoids have been removed the infection occurs in the lymphoid tissue in some other part of the pharynx, either in the lateral columns, at the base of the tongue, or low down near the pyriform sinuses. So that I believe that the laryngeal and tracheal abscesses, just as is the case in infections of the lateral pharyngeal columns, start as localized inflammatory processes, and then owing to the patient's run down condition, plus the addition of pyogenic organisms, the abscess gradually develops.

CASE REPORTS.

Case 1.—The first case I wish to report occurred during an influenza epidemic during winter of 1898, and was particularly severe in Albany and the surrounding towns.

The patient, a man, aged 34 years, was referred to me by Dr. Gordinier of Troy. Briefly, the history of this case, that Dr. Gordinier gave me, is as follows: Two months before I saw him he had a typical attack of influenza with a bronchopneumonia, which ran the usual course. He had the usual prostration that grip patients always have, after they have apparently recovered, but did not gain any strength and lost steadily in weight. His difficulty in breathing started about three weeks before Dr. Gordinier referred him to me. This became steadily worse, and the day he came to my office he was cyanotic and breathed with a decided inspiratory stridor. While I was examining him his breathing became so bad that I thought I would have to do a tracheotomy in the office. The laryngeal mirror showed the presence of a large, smooth tumor mass, starting at about the second tracheal ring and extending down as far as I could see. I rushed him to St. Peter's Hospital, as I had to lecture to a class of students there, and by the time we reached the hospital he had practically stopped breathing. We got him into the amphitheater and put him in a chair that could be tilted well back, and I performed an immediate tracheotomy. There was no time for even a local anesthetic. The incision through the tracheal wall was at once followed by a gush of pus and blood, which he coughed clear across the room, through the tracheal wound. Unfortunately in this case no bacteriologic diagnosis was made, and I did not suspect the presence of the abscess before operating. In the laryngeal mirror it looked like a tumor mass. He did very well for six or seven days, and then developed an aspiration pneumonia, from which he died on the thirteenth day.

Case 2.—Abscess of the ventricular band filling left half of larynx. This patient, a man, aged 31 years, had an attack of influenza during the epidemic of 1918. He had the usual symptoms in the beginning, with an intense sore throat, and an acute double frontal sinusitis. This condition cleared up under the usual treatment, but as in the first case, he remained very weak and lost weight steadily. His difficulty in breathing started about the third week from the onset of the attack, and about a week after I last saw him. His difficulty in breathing was quite apparent when he walked into my office, and on laryngeal examination a smooth mass was seen which filled the left half of the larynx and apparently originated in the ventricular band. I made a diagnosis of abscess, and under cocain anesthesia made a free incision with the old Schroetter guarded knife. There was an immediate free escape of pus, and he was placed with his head well forward, for a long time, so as to lessen the danger of aspirating any of the pus. Daily applications of 50 per cent alcohol were applied to the abscess cavity, which drained for about a week. He made an uneventful recovery and got his old weight back. Bacteriologic examination of the pus showed a mixed streptococcus and staphylococcus aureus infection.

Case 3.—Abscess of larynx, right side, also apparently originating in ventricular band. This case also occurred during the influenza epidemic of the winter of 1917-18. This case was so similar to the last one that I will not report it in detail. Patient, a man aged 31 years, had influenza during the epidemic mentioned above. He had an acute middle ear, left side, requiring incision, and an acute right maxillary sinusitis. This was opened through the inferior meatus and treated in the usual way. The abscess was opened as in case two and the cavity swabbed daily with 50 per cent alcohol. He also made a complete recovery. Microscopic diagnosis, mixed streptococcus and staphylococcus infection. Neither of the above two cases developed any bronchopneumonia.

Case 4.—Abscess on the posterior laryngeal wall, developing about the third week, right after an influenza attack, during the same epidemic. This patient, a man, aged 42 years, started with the usual sharp sore throat and acute rhinitis, without, however, any sinus or ear complications. On the fourth day of the attack he developed a slight bronchopneumonia, which cleared up in the course of a week. I did not see him for about ten days after this, when he came to my hospital clinic. He was breathing with great difficulty, and examination showed the presence of a large smooth mass on the posterior wall of the larynx. This was opened under cocain anesthesia, with the Schroetter knife, and proved to be an abscess. Examination of the pus showed a streptococcus aureus infection. He also made a good recovery, under the same line of treatment.

Case 5.—Abscess of the larynx, right ventricular band. The patient, a man aged 37 years, had a typical attack of influenza in January, 1918. He had no sinus or ear complications, but ran a very high temperature with great prostration, for over two weeks. He also had a diffuse bronchitis, but no definite bronchopneumonia. He was discharged, as apparently cured, on the eighteenth day, and I did not see him again for a week, when he came to the office complaining of difficulty in breathing. He lost weight steadily since the beginning of his influenza attack. On examination, the laryngeal mirror showed a smooth mass almost filling the right half of the larynx. A diagnosis of abscess was made and the abscess incised under cocain. Cultures and smears from the pus showed a mixed streptococcus and staphylococcus aureus infection. The abscess cavity was treated in the same way as in the other cases.

He made a good recovery, but got his strength back very slowly.

Case 6.—This case occurred during the grip epidemic of the past winter. I was called to see this patient, a man forty years old, late in December, by his family physician. He was just recovering from a sharp attack of grip, but for several days had been troubled with an irritating cough and very serious difficulty in breathing. The day I saw him he was quite cyanotic and there was a well marked inspiratory stridor. After spraying his lower pharynx with a 2 per cent cocain solution, a smooth globular tumor could be seen with the laryngeal mirror. This, as nearly as I could judge, started at about the third tracheal ring, on the right side, and almost filled the trachea. Right after getting back to my office I received an urgent call to come back to the patient's house. I found him breathing with great difficulty, and performed an immediate tracheotomy. A large amount of pus and blood was coughed out, through the tracheal wound, and cultures and smears proved a streptococcus aureus infection. He did very well for four or five days, when he developed a pneumonia, undoubtedly an aspiration pneumonia, and died on the eighth day following the operation.

In conclusion, there are one or two points worthy of note. The fact that no influenza bacilli were found in the pus is interesting. I have no doubt that they would have been found in the secretion from the larynx and trachea, in all the cases, early in the attack. The four laryngeal cases recovered, probably because the abscesses were high enough up in the air passage, so that the patient could constantly cough out the pus. It is a question whether the two trachea cases could have been saved if there had been time enough to have used Jackson's tubes, immediately sucking out most of the pus, after incision, but I am doubtful if it would have made any difference. The aspiration pneumonias develop simply from the constant flow and aspiration of pus into the bronchi, particularly the right bronchus.

LXXI.

SOME POINTS IN THE COMPARATIVE ANATOMY OF THE NOSE AND THE ACCESSORY SINUSES WHICH ACCOUNT FOR THE VARIATIONS IN THESE STRUCTURES IN MAN.

(LANTERN SLIDES.)

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In its simplest form the olfactory organ consists of two depressions in the integument of the snout which open externally but do not communicate with the mouth.

Fish.—The olfactory organs of the fish are two rather regular oval cavities separated from each other by a thick median wall or septum. Each of these cavities opens externally, and in some fish this external opening, or nostril, is divided and thus an anterior and posterior nostril is formed. There is, however, no communication between the nasal fossæ and the mouth. The septum supports a number of radiating processes extending into each nasal fossa. These processes are covered with olfactory mucous membrane in which the olfactory nerves end, and they correspond with the olfactory turbinals of higher vertebrates.

Dipnoi are fish which resemble amphibia in some respects. They live in shallow streams and bury themselves in the mud during the dry season. The nasal fossæ open into the mouth, and a very primitive lung is developed which functionates while the fish lives in the mud and no longer receives its oxygen through the gills. In this way anterior or external and posterior or internal nostrils are differentiated, and a free passage is formed through which air passes into the lungs and the olfactory organ becomes a part of the respiratory system.

Reptiles, the Turtle.—The character of the nasal organ is modified in reptiles, by the change in the method of respiration. The nasal fossæ open into the mouth and are differentiated into an olfactory and respiratory portion.

The olfactory organ of the turtle consists of two symmetrical cavities with a relatively thick partition in the median line. Each cavity is subdivided into a respiratory and an olfactory region. The respiratory part is a tubelike structure which exends downward and backward and opens into the anterior part of the mouth, thus forming the posterior nares. Around these two openings into the mouth there are numerous fingerlike projections which, when they are brought into apposition, close off the posterior nares from the mouth.

The olfactory part of each fossa is a round cavity, above the respiratory region, and partially separated from it by a prominence projecting out from the lateral wall. This prominence corresponds to the turbinal in other vertebrates.

In more highly developed reptiles the secondary palate is formed by the union of the palatine processes of the superior maxillary bones with the pterygopalatine arch. Thus a secondary roof of the mouth is formed distinct from the sphenoidal or primary roof. The cavity thus formed below the sphenoid bone prolongs the nasal chambers backward so that they open into the nasopharynx instead of the mouth.

Birds, the Duck.—In most birds the anterior portions of the nasal fossæ communicate with each other, the septum being incomplete, and posteriorly the two fossæ have a common opening into the pharynx, which is surrounded by fingerlike projections similar to those around the posterior nares of the turtle.

In the duck there are three turbinals in each fossæ. The anterior one is an inverted "T," attached to the roof of the nasal chamber, and is suggestive of the maxillary turbinal with a double coil seen in the higher forms of vertebrates. It is, however, covered with stratified epithelium and is called a pseudoturbinal.

There are two ethmoidal turbinals. The anterior inferior one has a double coil and occupies the greater portion of the olfactory part of the nose. The superior ethmoidal turbinal is a simple prominence with two irregular projections extending downward.

Mammals.—The turbinal structures in mammals exhibit great variations. In microsmatic mammals (animals with an acute sense of smell) the turbinals attain their highest degree of development and are very complicated structures.

Panther.—The turbinals in the American panther are a good example of this high degree of development. The nasal fossæ are separated from each other by a thin, straight septum which unites with the cribriform plate posteriorly. Inferiorly the septum is formed by the vomer and presphenoid, and superiorly by the descending plates of the frontal and nasal bones. Along the articulation of the perpendicular plate of the ethmoid with these descending plates a lateral process extends out at right angles into each fossa and supports a considerable portion of the olfactory turbinals and partially subdivides the olfactory portion of the nose into two superimposed cavities.

The maxillary turbinal is in the anterior part of the nasal fossa. It springs from the superior maxillary bone by a slender pedicle, which extends out into the fossa, nearly parallel to the floor of the nose, and divides into two branches. The superior branch curves upward, making one complete coil. The inferior branch curves downward and makes two turns within itself. The whole structure is a typical example of the double coiled turbinal, which is present in many mammals. It is the homolog of the inferior turbinal in man.

The nasoturbinal is a rather simple structure, which projects downward from the nasal bone and overlaps the maxillary turbinal and the anterior part of the ethmoid turbinals. Its homolog in man is the ager nasi, a small bony ridge anterior to the middle turbinal.

The ethmoid or olfactory turbinals are very complex structures. There are six of these turbinals in each fossa. They radiate from the convex surface of the cribriform plate, and the extent of their surface is tremendously increased by almost innumerable folds and branches, which make an intricate labyrinth occupying the posterior part of each nasal fossa and constitute the olfactory portion of the nose. Branches from these turbinals extend into all of the accessory sinuses.

The posterior portion of each nasal fossa is subdivided into two superimposed cavities by the lateral wings of the vomer.

The superior cavity is the olfactory portion of the nose, and contains most of the olfactory turbinals. The inferior cavity connects the respiratory portion of the nose with the nasopharynx.

Dog.—The maxillary turbinal in the dog is attached to the superior maxillary bone by a single shelflike process extending along the external lateral wall of the anterior portion of the nasal fossa. Numerous curved branches are given off, which nearly fill the anterior nares and present a very large surface of respiratory mucous membrane. The double scroll shape of the maxillary turbinal is supplanted by the numerous branches, except in the posterior inferior portion of the turbinal. Here the scroll shape still persists.

The ethmoid turbinals are very complex structures, consisting of many thin delicate scrolls and branches, of which at least five radiate from the convex surface of the cribriform plate. Some of the numerous branches of the ethmoid turbinals extend into all of the accessory cavities of the nose. One scroll is supported by a process which is attached to the external lateral wall of the maxillary sinus.

The maxillary sinus is hollowed out of the superior maxillary bone, and it communicates with the nasal fossa through a large opening which extends from the floor of the cavity to its roof. The process which springs from the lateral wall of the sinus and supports a part of the ethmoid turbinal extends into the nasal fossa through this wide opening. The frontal sinus is situated between the two plates of the frontal bone, and its anterior inferior part is occupied by a double coil of the ethmoid turbinal. The two sphenoid sinuses are separated from each other by a rather thick septum and are nearly filled by scrolls of the ethmoid turbinal.

The posterior part of the vomer is expanded into two lateral wings which unite with the ethmoid bone and divide the posterior part of each nasal fossa into two superimposed cavities. The superior cavity contains most of the ethmoid turbinals and is distinctly an olfactory chamber. The inferior cavity is a tubelike structure forming a direct communication between the anterior half of the nasal fossa and the nasopharynx.

The function of the accessory cavities of the nose in the panther and the dog is to provide space for the tremendous development of the olfactory turbinals, to conduct air over the external lateral surfaces of these structures and to furnish a system of drainage for them.

Apes.—The ape's nose is a very much degenerated structure. The maxillary turbinal is quite similar in outline and position to the corresponding turbinal in man. In some apes it has only a single inferior coil. and in others it still has a small superior coil also.

The ethmoid turbinals vary considerably. There may be only one or two or three present, and in a few apes a rudiment of the nasal turbinal shows quite distinctly. The numerous fine convoluted branches of the ethmoid turbinals which are present in lower animals and the fourth and fifth ethmoid turbinals have all disappeared.

The maxillary sinus is usually fairly large and opens into the middle meatus through a small ostium. The walls of the sinus are smooth, and the sinus contains no ethmoid turbinal tissue. Usually in apes there are no frontal sinuses, and the frontal bone is cancellous. The chimpanzee, however, has two fairly well developed frontal sinuses. The ethmoid cells vary in size and number. In some apes there are none and the ethmoid turbinals are very rudimentary. In others, there are several ethmoid cells, and the ethmoid turbinals are more highly developed.

The sphenoid cells vary also and are present in those apes which have ethmoid cells. and in the orang-utan the sphenoid cells show rudiments of ethmoid turbinals.

Man.—The general characteristics of the accessory cavities of the nose in man are too well known to need a description here. They show numerous variations, many of which are reversions to more primitive types. The absence of one or both frontal sinuses corresponds to the absence of these sinuses in most apes.

The ethmoid cells are formed by the union of the numerous branches of the ethmoid and adjacent bones and occasionally show small irregularities which suggest rudiments of the ethmoid turbinal scrolls which are well developed in some of the lower animals.

The subdivisions of one or both sphenoid sinuses correspond to a similar division of these sinuses in some of the lower animals in which the ethmoid turbinals extend into the sphenoid sinuses and thus produce numerous subdivisions. The irregular ridges sometimes present on the lateral wall of the maxillary sinuses are probably rudiments of the ethmoid turbinals which in some animals extend directly into these sinuses and are supported by attachment to the external lateral walls of the sinuses.

The accessory sinuses in the lower animals develop in connection with the olfactory portion of the nose and form channels which conduct the inspired air over the external lateral surfaces of the olfactory turbinals and in this way increase the acuteness of the sense of smell. In the higher orders of mammals, particularly the apes and man, the olfactory turbinals have degenerated and receded from the accessory cavities, and the cavities are left with comparatively smooth walls, and their functional activity is lost.

In man the accessory cavities are almost completely shut off from the nose. Their functional activity ceases and they remain as rudimentary structures.

LXXII.

INTRANASAL INJECTION OF ALCOHOL IN THE TREATMENT OF HYPERESTHETIC RHINITIS AND SOME OF THE NASAL NEUROSES.*

By Otto J. Stein, M. D.,

Chicago.

In considering the treatment of any disorder one can approach the subject from several angles. In the case of the neuralgias of the head, this approach can be in the form of a direct attack upon the nerves involved, either by injecting them or by a surgical resection. With the vasomotor disturbances of the nose one can approach the subject in a like manner, although it is essential at all times, as it is in neuralgic cases, to give due importance to individual susceptibility or sensitization, causation and differentiation. Knowing the cause and removing it or correcting the patient's susceptibility may be sufficient to relieve him of his trouble. While this may be done in some cases, the difficulties surrounding these methods are much greater than would appear from the many excellent articles that have been published of late years on sensitization and deficient metabolism, and the favorable results are far from what the alluring but grossly misleading advertisements from overzealous manufacturers would have one believe.

It is assumed that a searching survey of the nasal chambers in every instance should precede any radical method of treatment. Therefore, shrinking of the tissues and anesthetization to permit inspection with the Holmes electroscope, plus transillumination or roentgenograms is good practice. A suppurating cell, hyperplastic sinus, a sharp spur, an ulceration, an adhesion, any of these may enter into the etiology of nasal neuroses. It may require a cutaneous test to differentiate them from true "hay fever."

In my first presentation of this subject, which was in a paper read before the American Academy of Ophthalmology and Otolaryngology in 1907, I proposed a method of treating these disorders by a direct attack upon the intranasal nerves. This work was commenced in 1906, following the suggestion of Schlosser in the treatment of neuralgia and of Killian for anesthetic purpose. I am not aware of this method of treatment having been employed for these neuroses prior to this time, although, as I just said, it was being used for the relief of painful impressions, both local and reflex in character. Since my introduction of this method of treatment I have persistently followed its principles in the greater number of cases of this class. I have also presented several additional papers on the same subject (The Section of Laryngology and Otology, A. M. A., Chicago, June, 1908; Interstate Medical Journal, Vol 17, No. 7, 1910; Reference Hand Book of the Medical Sciences) and have instructed numerous physicians in the technic, and who are using the method right along. Any changes from the original in instrumentation, method of approach, selection of cases, results, complications and the like that may have occurred since this work was first presented I will reserve for discussion until the latter part of my paper.

It is a well recognized fact that the great fifth nerve and its intimate connections with the sympathetic and motor nerve systems plays an important role in a variety of disturbances arising as a result of its stimulation or irritation, whether this takes place from within or from without. The immense area of its peripheral distribution to the mucous membranes of the nose, throat and eyes subjects it readily to untoward influences, such as may act as irritants or excitants that in turn create reflex manifestations that can be grouped into various symptoms complex. It is not necessary to enter into a descriptive recital of the anatomy of the fifth nerve, but sufficient should be said to show how these reflexes may occur. Its sensory root is said to anastomose with all motor nuclei, with the exception of the sixth nerve, coming from the medulla. In addition, the fifth nerve receives sympathetic fibers from both the carotid and cavernous plexus, the latter going to the ophthalmic division and its branches and possess vasomotor function. The sympathetic fibers from the carotid plexus enter into the makeup of the sphenopalatine ganglion (Meckel) as the large deep petrosal nerve. Motor fibers from the facial nerve also enter into this makeup through the large superficial petrosal nerve. These two petrosals, transmitting motor and vasomtor impulses, as well as sensory and perhaps secretory impulses, unite under the name of the vidian nerve.

The sensory element of this (Meckel) ganglion is derived from two sphenopalatine nerves branching off from the superior maxillary or second root and with that of the nasal nerve derived from the ophthalmic or first root constitute the sensory source of the nasal membranes. As the different fibers leave the ganglionic area for distribution to the membrane lining the nasal chambers and accessory sinuses, they convey all the impulses that go to make up the complexion of the ganglion, namely, sensory, secretomotor, vasodilator, vasoconstrictor and probably trophic. In this way we can account for the tickling and itching sensation in and about the nose, eyes, throat and roof of mouth; the sneezing, the stuffiness, the flow of serum, mucus and lacrimal fluid and the respiratory embarrassment, all the result of stimulation or irritation of these peripheral nerves. I do not think it possible for me to enter into any discussion on the intricate physiologic activities of this ganglionic area because the subject is so complex and so beset with unsolved problems that much is as yet speculative. Just why, after an irritation of these nerves, there should follow in one instance pain, in another reflex asthma, in another rhinorrhea, in another the so-called hay fever syndrome is a physiologic anatomic study that is not within my province at this time, but it nevertheless opens up an immense field for speculation and investigation. One might formulate a law just as Semon did regarding the paralyzing effects of the recurrent laryngeal nerve in which abductor fibers take precedence to the adductors.

For the purpose of simplifying the understanding of this method of treatment, it is usual for me to divide the distribution of the intranasal nerve supply into two main divisions, the anterior and posterior. The anterior division is the nasal nerve, sometimes called ethmoidal. It is one of the three large branches of the ophthalmic root. The other two branches are the frontal and lacrimal. The frontal, being the main branch, terminates as the supraorbital and supplies the upper eyelid, brow, forehead and temples. The lacrimal supplies the lachrimal gland and the upper lid. The nasal nerve branches off from the ophthalmic root near the sphenoidal fissure. It passes across the upper part of the orbit diagonally forward from the outer to the inner side to pass through the anterior ethmoidal foramen into the ethmoid cells and, after giving a branch to the frontal sinus, it leaves the ethmoid and enters the cranial cavity at the outer border of the cribriform plate; it then passes forward to a slit at the base of the crista galli, which it enters, emerging into the anterior superior angle of the nasal attic chamber about a millimeter or two lateral to the septal articulation. This is the location of election for reaching the nerve intranasally for the purpose of blocking. After it enters the nose a septal and external branch are given off. These branches supply the anterior part of the nasal chamber, also the ala, bridge, tip of nose, brow and forehead. The posterior divisions are branches arising from the sphenopalatine ganglionic area. Through these various branches the posterior nasal chamber and all accessory sinuses, excepting the frontal, are supplied. Filaments are also distributed to orbit, palate, nasopharynx, mesopharynx, eustachian tube, etc.

One of the reasons why certain patients are afforded only partial or no relief from their nasal neurosis after they have had spurs removed, septum straightened, turbinals cauterized and sinuses irrigated is because only a few of the many different nerve filaments have been reached by such procedures. It is well enough to correct such gross irregularities if the symptoms are localized, but where the irritation is of wider distribution then, in addition or separately, the area at the point of nerve entrance into the nose should be blocked. The method of doing this is as follows: For the purpose of injecting the anterior division of the nerve, a straight steel needle of about 22 to 24 gauge and 9 cm. long with a slip joint to securely fit an all-glass syringe is used. This fitting has a marker on it indicating the side of the bevel at the other or pointed end. This point should be sharp, but at the same time of short bevel, in order to lessen the possibility of fluid escaping. As the point of the needle is only introduced to a depth of about a mm., the bevel should not be any longer than this. After previously preparing the field for injection by shrinking and anesthetizing with a solution of cocaine and adrenalin, the straight needle is fixed to the syringe containing the alcohol in such a manner as to show by the indicator on the slip joint that the bevel point is directed forward, and the instrument so prepared is carried to the foramen by following closely the septal wall until the vault is reached, a distance of about 5 to 5½ cm. from the nasal crest; then the point is carried slightly outward as the slit through which the nerve enters is about 1 to 2 mm. lateral to the septum. If properly placed, the point of the needle can be felt entering the opening. No force is necessary. On some occasions I have felt the needle entering the opening for some considerable distance, although this is not necessary or advisable. It is sufficient that the bevel of the point is buried in the foramen so that no fluid escapes into the nostril. The neighboring nostril is injected in like manner. Variations in the depth of the floor of the frontal sinus in only a few rare instances change the location of the foramen sufficiently to warrant more than mentioning. Very high deflections and old fractures offer the greatest obstacle to accuracy. To overcome this I use a very fine and somewhat flexible needle that will pass around the obstruction. The easiest technic is to hug the septum and follow directly beneath the nasal bone. The external branch of the nasal nerve lies in a groove or at times a tunnel in this bone. This branch is of less importance than the inner one, but both are secured at the orifice of the foramen. The distances are slightly altered by age and types of heads. That is to say, in young children a shorter and finer needle should be used. The needle used for injecting the posterior division is 10 cm. long, 16 to 18 gauge, with slip joint and indicator showing the location of the bevel at the point. This point is sharp and of short bevel and bends at about a right angle from its shaft for a distance of about 10 mm. For the very wide nostrils and deeply located fossa a bend of 15 mm. is used, and in like manner one of 7 mm. for closely fitting turbinal and septum. After preliminary cocainization and thoroughly shrinking of the tissues, carry the curved end of the needle to the posterior nostril, hook it gently around the tip of the middle turbinate, and at a point where this body arises from the lateral wall will be found the foramen of the sphenomaxillary fossa. Introduction of the needle point into the membrane covering the foramen will reach the nerves emerging therefrom. If the point of the needle is carried three or four millimeters posterior to the first position and then forced laterally through the membrane, a more accurate penetration of the ganglion is possible and with less chance of injuring any vessels. A forward projecting sphenoid or peterygoid process may make this difficult, as Sluder has pointed out.

In injecting the posterior group of nerves, I advise selecting the region of the sphenopalatine foramen as the point of election in which to introduce the curved needle point. It is true that the nerves leaving the spenomaxillary fossa do not all make their exit at the foramen, only the posterior superior nasal laterals, that supplies the correspondingly named membrane, and the nasopalatine nerve that supplies the vault and septum. The others leave by other exits to enter canals that lead to all the neighboring parts. But one must not lose sight of what the histology of the gland teaches, namely, that many of the sensory fibers from the second root do not even enter into the complexion of the gland structure, but pass directly on to the nasal chamber and adnexa. It is to be remembered that the ganglion usually lies directly against or at least very close to the foramen and can be reached, in part at least, by proper placement of the needle point. But aside from this, an injection at the foramen reaches those nerves supplying that part of the nasal membrane where most of the protein substance is likely to be found. Probably it is necessary in some cases, as Sluder has said, that in order to reach the ganglion itself and thereby effect a desensitization of all functional elements contained in the fossa, the straight needle introduced trans middle turbinal posterior tip be used; but in the type of cases under discussion I believe the curved needle is preferable and fully satisfactory. As a matter of fact, my experience has been that only a few of this class of cases require the posterior injection, especially where an accurate anterior injection has been negotiated, and provided the treatment is instituted early. When one is satisfied the needle does not accurately enter the desired place, another attempt should be made in a few days, or where the benefits are only partial after anterior injection a posterior injection may be added. The relief is usually for the season in hay fever cases. In simple hydrorrhea, spasmodic coryza and nasal reflex asthma it usually acts instantly.

Alcohol is a most suitable substance for injection, because it is sterile, nontoxic and noncorrosive. It has been used extensively for the purpose of injecting nerve tissue, and it records no particular untoward results. Its action on nerve substance is a disorganization of its cell elements, causing some hardening but no necrosis. Otto May's investigations on the changes that take place in ganglionic cells and nerve fibers by alcohol (British Medical Journal, Aug., 1912) show the nerve fibers to be much easier affected than the cells. The functional activity of the nerve is restored to normal within a variable period, as is shown by the necessity for reinjecting each season in the seasonal cases. The more exact the injections are made the more effective and lasting are the results. Infiltration of the membrane in the neighborhood of the foramen is far less effective, but one does secure some immunity. Like any similar injection, a careful sterilization of instruments and field and a slow introduction of the fluid is essential to best results. Originally I used absolute alcohol but found this more painful than a 75 per cent dilution with fresh sterile water that I now use. About 10 minums is used for each injection. If the nerve is properly located a sharp pain radiating in the direction of the nerve distribution is immediately experienced on introduction of the fluid. This may last for some time unless anticipated by a previous hypodermic of morphin and atropin. When the procedure is carried out carefully and accurately in otherwise normal individuals there should be no serious or damaging results following. Sometimes a prolonged pain follows the injection, particularly when several areas have been entered or where the alcohol may have entered the cranial cavity and irritated the meninges; but this can be combatted with morphine. Where an infiltration results instead of a block a temporary anosmia may follow the anterior injection. Sluder records one instance of abducens paralysis of temporary duration following his posterior method. Serious bleeding has never occurred with me when the curved needle was used. Most of the sufferers are nervous with little self control and often restless.

In such patients the nasal membrane during an attack is exquisitely sensitive, so much so that even psychic impressions are sufficient to excite vasomotor and secretory activity. For this reason it is best to administer morphin and atropin prior to the treatment and a tablet containing a small dose of codein and caffein at intervals of a few hours following.

LXXIII.

THE G. E. BARNES DRUM MEMBRANE KNIFE AND SPATULA.

By George E. Barnes, B. A., M. D.,

Herkimer, N. Y.

There are certain objectionable points in the construction of myringotomes which have been on the market to date. The length of the blades is greater than advisable for incising the thin membrana tympani or even the posterosuperior meatal wall. The curves of the blades, whether convex or sickle shaped, are not only not required but actually unadapted to their work in such a small canal: during incision of the membrane, particularly from above downward, the long convex blade may cut the meatal wall; and during incision of the membrane either upward or downward the sickle shaped blade, because its curved back touches the meatal wall and thus prevents proper placing of the point, begins the incision too far from the margin of the membrane and tends to carry it beyond the desired spot at the terminal end. Furthermore, with a curved blade the operator cannot accurately estimate where its point is and, therefore, he may possibly insert the blade through the membrane farther than intended and do injury, particularly to the incudostapedial articulation.

To avoid these objections and to get an instrument that is adapted to its purpose as nearly as possible I have had made and wish to introduce the myringotome illustrated. The blade is straight, 1 mm. wide, 5 mm. long on its cutting edge, the point being formed by removing, in a somewhat oval line, the last 2 mm. on the back. It is thin, highly tempered and inflexible, and well sharpened.

The advantages of this blade scarcely need mention. Its short, straight cutting edge is long enough to execute the desired incisions and will not injure the meatal walls and its point, it being in line with the shank and the cutting edge, can be accurately placed wherever desired and its position in the middle ear during the act of cutting can be quite accurately estimated. The blade is made for its work.

The shank next to the blade, for 2.5 cm., is very slightly less than 1 mm. in diameter and for its other 5 cm. it gradually increases up to 2 mm. in diameter at the bayonet offset. The total length of the blade and shank is, therefore, 8 cm. or a little more.

The shank, unlike that of most myringotomes, is long enough to permit its use through an electrically lighted otoscope as well as through an ordinary ear speculum.

The bayonet offset is made with a distance of about 12 mm. between the adjacent surfaces of the shank and handle. This offset entirely removes the fingers from the line of vision.

The handle is 7.5 or 8 cm. long and about 4.5 mm. in diameter. It is hexagonal so that it can be held when desired without rotation and yet it can be easily and smoothly rotated between the thumb and index finger in describing the necessary arc with the bayonet handle in making a curved incision in the membrane.

The spatula differs from the myringotome only in the blade. This is 1 mm. wide and 5 mm. long and its end is quite thin but not sharp, the corners being merely smoothed off.

I introduce this spatula for the purpose of gently inserting it and drawing it the length of the incision when the lips of the incision become adherent, according to my ideas set forth in my article on the treatment of otitis media without irrigations or wicks in this journal, March, 1922. Its end should be passed in merely far enough to accomplish this and not the slightest unnecessary spreading of the lips should be made in order to avoid the entrance of fresh infection and the disturbance of the anatomical parts.

In order that these twin instruments may have some distinguishing feature when their blades are wrapped in cotton I suggest that the handle of the spatula be made somewhat flattened from side to side instead of round.

The illustrations show the whole instruments in half size and the blades in full size. They appeared in the March number of this journal.

SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of March 6, 1922.

THE PRESIDENT, DR. ROBERT SONNENSCHEIN, IN THE CHAIR.

DR. JOSEPH C. BECK presented a pathologic specimen of a

Lipoma of the Laryngopharynx,

which he had removed from a boy of sixteen. The tumor of the larynx had persisted for more than a year and had reached the pyriform fossa. The glands were not involved, the tumor was soft and not painful. The mucous membrane over it moved quite readily, and there was no pulsation. The trachea was enlarged, but only a droplet of material could be forced out and this showed evidence of fat. A piece was excised for microscopic examination, which revealed a true lipoma. No operation was suggested because they wished to try radium therapy. Three needles of radium were inserted but no effect was obtained, which verified the usual findings, that lipomata are not affected by radiotherapy.

A year later the patient returned with the growth considerably sunken down and the history of loud breathing and snoring during sleep. The growth was then enucleated under synergistic anesthesia by the suspension laryngoscopy method. Dr. Beck did not think he could have removed it in any other way, as the removal was somewhat difficult and he was compelled to cut it out with scissors.

Microscopic examination of the tumor had not yet been made, as the operation had occurred on the morning of the presentation.

DISCUSSION.

DR. HOLINGER asked the relation of the tumor to the tonsil and to the eustachian tube.

DR. BECK stated that the growth was back of the posterior pillar and did not extend beyond the margin of the soft palate. It was entirely from the nasal fossa.

Dr. C. M. Robertson said that a year and a half or two years ago a patient had come to him with stenosis from a laryngeal growth. He discovered some forty-odd fatty tumors over various portions of the body and one about the size of that removed by Dr. Beck was in the buccal cavity. Laryngeal examination showed a mass occupying the right side of the larynx. It did not look like a carcinoma and he suspected a lipoma owing to the number of lipomata distributed in other parts of the body. The patient was dyspneic, so it was necessary to do a preliminary tracheotomy and before they could prepare for an evisceration of the larynx he succumbed. Postmortem examination showed that the growth in the throat was a true carcinoma, although the other tumors in various parts of the body were lipomata.

Dr. Albert H. Andrews read a paper on

Surgery of the Ethmoid Labyrinth.*

*See page 947.

DISCUSSION.

Dr. C. M. Robertson agreed with Dr. Andrews concerning the anterior ethmoid cells and preservation of the turbinate. The type of case he recited was the one in which the ethmoidal cell is diseased and the turbinate is in place, and Dr. Andrews had stated that it was necessary to displace the turbinate inward at times in order to get at the ethmoidal cells. Many cases are seen in which new cells are forming, with ethmoidal cells extending into the middle turbinated body (cystic degeneration), and these cells push the turbinate over against the nasal septum, as so often seen in cases of nasal hydrops. Dr. Robertson had seen many cases in which the patient had been advised to have a submucous resection on account of the deflected position of the septum on the opposite side.

Dr. Robertson said he did the same operation as Dr. Andrews by a little different technic. After the labyrinth is cleaned he comes forward to get the anterior ethmoidal cells and then fractures the ethmoidal plate outward.

One case of pansinusitus was particularly interesting. It had now been five or six years since the operation was performed and the patient apparently had a normal nose so far as the size and position of the middle turbinate was concerned.

In that case the ethmoidal was pushed over a fourth of an inch and after the fracture of the bony plate the resiliency of vertical plate of the septum brought it back into a normal position. He considered this the most satisfactory case he had seen. There is now a normal turbinate carrying out the physiologic function, occupying its normal position with the ethmoidal space on the outside wall of the nose.

Dr. Edwin McGinnis thought many of those present knew how he felt on this question, and he was very glad to hear others talk of ventilation instead of exenteration. He has seen two cases of exenteration of the ethmoid this winter in which this operation had been performed four or five years ago. One of the cases was very interesting in that during every cold spell the girl would have a Sluder syndrome—headache, pain in the side of neck, tenderness back of ear, and inability to work. His explanation of this was that the cold air had free access to the region of the sphenoganglia, and this was proved by the absence of symptoms when that side of nose was plugged with cotton. Last summer she had no trouble whatever in the place where the ethmoid used to be.

Dr. McGinnis was interested in Dr. Robertson's remarks. Everyone in the last year had talked about the hyperplastic ethmoiditis. This operation under discussion is useful in the class of cases where there is no pathology in the ethmoid but much trouble bacteriologically. He had seen a pronounced case of asthmatic bronchitis recently in which the patient, a man, had great difficulty in breathing. He had to sit up and most of the time had to lean over a chair. It was not a hyperplastic ethmoiditis, but a bacteriologic affair. He made an incision in the bottom of the ethmoid cells, opening up the frontal, and found a staphylococcus aureus hemolyticus infection. This patient went along fairly well until he caught cold about a week after the operation. He then had quite a stormy time for about a week. Dr. Carey grew the staphylococcus hemolyticus and injected rabbits. One rabbit died within a week with pneumonic areas through the lung, Villous pericarditis, acute nephritis, and one died inside of another week. The third rabbit was still alive. At times there have been albumin and casts in the urine.

Many cases of ethmoiditis have to have something done for them and many of these patients will not submit to an exenteration, but the ventilation will be of much benefit to the patient. The man referred to had an apparently normal nose that showed no evidence of having been operated upon. He had a bad asthmatic attack a week following operation, but had been very comfortable since that time, and was sleeping well at night. His color was good.

Dr. McGinnis thought Dr. Andrews missed one point; it is impossible to get at the ethmoid in some cases unless one takes out a part of the septum high up, particularly with a broad deflection of the septum.

He had used no vaccines in his case. In his opinion it did no good to operate and then give vaccines or potassium iodid, for one should strive to get a good, positive result with operation and nothing else.

Dr. Frank Brawley thought everyone was indebted to Dr. Andrews for stressing the point of conserving the turbinates. and supported Dr. McGinnis' remarks concerning the Sluder syndrome. He had an exactly similar case which he proved by placing cotton in the nose during an attack, with complete relief. Dr. Brawley believed the profession now realized more thoroughly what the middle turbinate was placed there for, and were thus avoiding unpleasant complications.

Dr. H. W. Loeb thought the keynote was expressed in the last sentence in the paper—"We can never put back what has been removed, but can always remove more." He had not been able to save the middle turbinate quite so often as had been brought out by the speakers, nor did he think this was necessary. Dr. Loeb believed anything that would make operators more respectful of the normal tissues about the nose was of value.

Dr. A. H. Andrews (closing the discussion) said that some years ago he devised an instrument for removing turbinates that was said to make the operation so easy that when men owning the instrument did not know what to do for a patient they removed the middle turbinate. If this was true he thought he had something to answer for in the future. Dr. Andrews wanted those present to remember that whenever the middle turbinate and ethmoids were removed that the nose was forever a crippled nose. The removal may be necessary, regardless of future results, but it always leaves the function of the nose impaired. Whenever an air filled cavity, such as the nasal accessory sinus, becomes closed off from the outer atmosphere, the cavity always becomes diseased. The negative pressure which results from lack of ventilation causes an increase in the blood supply, an exudate, and sometimes causes granulation tissue to form in the cavity regardless of the class of microorganism. In these cavities the object sought is ventilation and not excision or drainage.

Dr. Andrews thought it was sometimes necessary to operate on the septum in order to get at the turbinate, but this was not included in the scope of the paper. In his cadaver work he had never found ethmoid cells which were entirely supraorbital; the supraorbital cells are merely instances of the ordinary cells extending out over the orbit. When the ventilation is secured for the ordinary ethmoid cells the supraorbital cells are also provided with both ventilation and drainage.

DR. W. E. GROVE, Milwaukee, Wis., read a thesis entitled

Mishaps in the Puncture and Irrigation of the Maxillary Sinus.*

*See page 913.

DISCUSSION.

DR. A. H. ANDREWS emphasized two points, one touched upon by the essayist. After opening the antrum through the canine fossa he had pushed the trocar through the inferior nasal antral wall while observing the result through the canine fossa. In a large proportion of cases the mucous membrane would be pushed ahead of the trocar. If this happens in the living, when the trocar is removable and compressed air turned into the canula, if the patient does not die he should be expected to.

A point which Dr. Andrews did not hear mentioned by the essayist was the means of exit for air or fluid. Whenever one makes a puncture and turns air into the antrum without providing abundant opportunity for its escape, he is causing great discomfort to the patient and unnecessary risk to his life. Dr. Andrews' impression was that if the operator is careful to see that the trocar and canula are in the antrum instead of between the mucous membrane and the bone, and that there is abundant means of exit for whatever is used, whether air or water, such unfortunate results as had been reported tonight will be entirely avoided.

Dr. Joseph C. Beck said that he never inflated the antra and had always felt it was a very dangerous procedure, although he knew that excellent men were doing it. He could not speak of mishaps from that procedure or any other, but instead of washing much he uses the negative suction after puncturing the antrum. In this way he obtains all the desired diagnostic and therapeutic results, and agreed with the statement that there was no harm in a little medication in the antrum.

The point made by Dr. Andrews he considered very important. If he had to puncture through the inferior meatus he never neglected to see that the middle meatus was open. It was sometimes very difficult to go through the natural opening and thus it became necessary to go through the middle meatus.

Dr. Beck thought Dr. Grove's paper was very opportune because of the question of the use of negative pressure instead of the washing or the use of air. At a recent discussion on this subject before the Clinical Society in the hospital Dr. Orndoff had spoken of the use of pneumoperitoneum and air in the ventricles for diagnosis. Surgeons are using this method for the diagnosis of other lesions about the joints and about the kidneys, and Dr. Beck wondered if air could not be used for diagnosis in the laryngeal field. In the discussion it was stated that they were afraid to get the air into the soft tissues, not necessarily into the blood vessels. In the case of air in the orbit, mentioned by Dr. Grove, which affected the optic nerve, this could be explained by getting the air around the periosteum, around the orbit and locking it into the optic foramen, causing blocking and secondary reaction, or infection, around the nerve. Dr. Beck had seen two cases in his clinic in which his assistants had punctured through the antrum, evidently through the wall of the sinus, and got an emphysema in this way. He thought this accident was quite common.

Dr. Beck considered Dr. Grove's paper one of the best the Society had had the pleasure of listening to, and thought the doctor had done a great amount of work.

Dr. H. W. Loeb congratulated the Society upon having such a well studied paper. As he did not puncture the antra he felt that he could not add anything to the paper. He was much interested in the fatalities reported and said he would be glad to make use of Dr. Grove's statistics in a paper he had under preparation.

Dr. Frank Brawley reported two cases of syncope, possibly produced in the way Dr. Grove reported. Neither one occurred following puncture, but both in cases where numerous irrigations had been made. One was in an antrum and one in a frontal sinus and both occurred following insufflation with air. He had never had one follow a sinus puncture, probably because he used a very sharp instrument for the first step and then a blunt canula so that before the insufflation he knew there was plenty of space for return and a freely movable canula in the opening. Neither of these cases was serious; there was very little change in the pulse rate, nothing but the syncope and stertorous breathing. In one of the cases an intranasal frontal sinus operation had been done the year before. There was no difficulty in finding the opening but following irrigation, when the air was insufflated, the patient in about thirty seconds complained of feeling queer and immediately passed into syncope. A diagnostician examined her at the time but found nothing. It was about forty-eight hours before she fully recovered and she was semiconscious during this period. The fundi was examined several times, but nothing was found despite the fact that she complained of loss of vision during the first thirty minutes.

Dr. Alfred Lewy said that in the large majority of cases where it was desired to investigate or to treat the antrum it was not even necessary to subject the patient to the trauma of puncture, as it was possible to enter the antrum through the natural opening in over 80 per cent of cases. Dr. Skillern has stated that in less than 10 per cent was it possible to do this, but Dr. Lewy and several other members of this society that he knew had been doing this for many years, and he believed the statement that it could be done in 80 per cent of cases was conservative. He had performed this little treatment about 2000 times and only once had he entered the orbit, causing a transient swelling of the eyelids. He used the little canula which is credited to Dr. Pierce, but which Dr. Pierce ascribes to Sir Morrell Mackenzie. It has two openings on the side, with a smooth, blunt end, and under proper manipulation seems to find its way into the natural ostium, so that in a majority of cases there is not even a drop of blood drawn by the procedure. Patients who have been treated by this method and by the puncture method have no hesitancy in making a choice.

Dr. John A. Cavanaugh reported two mishaps, one in an antrum case and one in a sphenoid. The antrum was punctured by an assistant in the inferior meatus and compressed air used to blow out the cavity. This patient became pale, complained of inability to breathe freely and claimed that it was ten days before he could breathe properly again. He felt as though his chest was in a vice, but he recovered and progressed nicely.

In the sphenoid case a canula was passed through the natural opening and air introduced very gently. The patient fell to the floor and became cyanotic. Dr. Cavanaugh could not account for this symptom, until he opened the mouth and found a plate which had fallen back and obstructed the respiratory passage. When he removed the plate the patient revived.

Dr. Cavanaugh had recently seen a surgeon wash the antrum by introducing a needle underneath the inferior turbinate, following the methods seen in textbooks, placing the hand on the side of the head and giving the needle a shove. Dr. Cavanaugh considered this poor technic. It is well known that the antral wall is very thin and if one takes hold of the needle about a fourth of an inch from tip of nose after the needle point is placed under the inferior turbinate there is no danger of mishap. In inserting the needle with a shove one is likely to go through the antrum into the orbital cavity.

Dr. Robert Sonnenschein corroborated what Dr. Lewy said and stated that for five or six years he had very rarely punctured an antrum. He had also had the little procedure performed on himself several hundreds of times, first by Dr. Pierce and later by Dr. Lewy. In most instances if a little cocain is placed in the middle meatus the operation is practically painless and he had never experienced an unfortunate effect.

Dr. Sonnenschein stated that he had had the pleasure of reading Dr. Grove's paper in its entirety and that it was a most thorough and excellent piece of work. He thought the Society was to be congratulated in having it presented.

Dr. W. E. Grove (closing the discussion) said that he had failed to emphasize the point brought out by Dr. Andrews regarding the removal of polyps from the middle meatus and having this patent.

Dr. Beck had spoken of suppuration, and if Dr. Grove had read his paper in full this would have been taken up. The blocking at the optic foramen was one of the first things that had occurred to him, but he could not figure how the air could strip all the mucous membrane up and account for the blocking. He had recently had a patient who was skating and was struck in the upper lid by the point of a skate. The skate penetrated his upper lid and evidently gouged out his eye. Two days later he could get this open and the eye ground presented a remarkable picture; the veins were absolutely black, the retina was quite pale and in the course of the last two weeks the disc had become atrophic. Dr. Grove thought a certain amount of hemorrhage had been produced postbulbar which shut off the return circulation long enough to cause a devitalization and optic atrophy.

Kümmel had reported two cases where he inflated the frontal sinus and Moritz Schmidt has also reported inflating the frontal sinus with air.

The points brought out by Dr. Lewy were covered in the part of the paper which he had not taken time to read. He called attention to the fact that sounding the normal opening was open the posterior fontanel of the middle meatus. The bony lamella is so thin that one can push in with a blunt canula, thus getting in very easily and often instead of sounding the opening one is making an actual puncture in the fontanel.

With regard to the thickness of the ethmoid wall, Onodi has called attention to the fact that it sometimes reaches a thickness of 3 millimeters, especially in the lower portion, near the floor.

Dr. John Alfred Rundstrom presented an inaugural thesis entitled:

The Pathologicoanatomic Difference Between the Fetid and the Nonfetid Ozena.*

*See page 950.

DISCUSSION.

Dr. S. M. Morwitz said that last year he spent six months in the clinic of Prof. Hajek at Vienna. Here Dozent Hofer next in rank to the Professor, obtained his title through his researches on ozena, and he felt convinced that the origin of ozena is entirely bacteriologic and its treatment serologic by virtue of an active immunity produced. He had prepared a vaccine called vakzine coccobazillus foetidus ozenae. He gives intramuscular injections weekly beginning with 50,000,000 in 1 c.c. and increases to 500,000,000 in 1 c.c. Twenty to thirty injections are given, all told. The patient was examined two or three days after each injection to note any reaction in the nose, at point of injection, or for constitutional symptoms. This form of treatment is only of value in the socalled early type cases, not in the well advanced ones, although sometimes the odor will disappear in the advanced type. Furthermore, a patient must be in good physical condition to receive this kind of treatment. Dozent Hofer claims wonderful results from the use of his vaccine. I examined repeatedly a number in his series of cases and the results appeared encouraging. He also demonstrated several specimens of turbinates in rabbits which had been injected with the coccobazillus foetidus and in which a definite atrophy of the mucous membrane of the turbinates had been produced. In Berlin Prof. Halle employs a surgical method in ozena cases. He displaces the lateral nasal walls towards the septum thereby reducing the air space of the meati. He is enthusiastic about the results thus obtained.

Dr. Robert Sonnenschein asked if this bacillus was not the same one that was described several years ago by Perez of Buenos Ayres. If so, it was nothing new.

Dr. W. E. Grove was interested by the fact that ozena is more prevalent in certain parts of the country, in some places there being none at all and it being very prevalent in others.

In his work with Gerber in Koenigsburg they found that in many localities there were many cases, while in other places there were none at all.

Dr. J. Holinger thought the paper showed a great deal of work, microscopic as well as bacteriologic, and that it was one more contribution to the theories that ozena is originally a disease of the sinuses. So many different theories were finding their support. The French at one time were absolutely sure they had found the cause of ozena in a special kind of diphtheria bacillus and gave antitoxin until it was shown that the results, if any were obtained, were short lived. The question of the sinuses being the cause had been advanced for more than twenty-five years and the feeling of raw bone at the entrance of any or several of the sinuses was given as pathognomonic. Dr. Holinger had many times tried to feel raw bone with a probe and while in one or two cases he may have succeeded he could not convince himself in all cases. He thought the greatest importance for the explanation of ozena laid in those cases where one finds typical ozena on one side of the nose and normal conditions on the other side. If one would ascribe the origin of the disease to a definite microorganism, it was almost impossible to believe that this microorganism could exist in one side and be absolutely absent in the other.

The question of geographic distribution and racial predilection may coincide with the idea of those who say that ozena patients have from the beginning a nose that is wider than normal. If to this is added a chronic suppuration in childhood, sustained for years by poor sanitary conditions we may expect changes of the lining of the nose, and Dr. Holinger believed two things were essential: a too wide nose and a metaplasia of the nasal epithelium on the middle turbinate. This metaplasia may be caused by old suppuration of the sinuses. He believed the question will remain unsettled, and while he congratulated the Society and the reader of the paper upon the thorough work that had been produced, on the other hand, he would not be surprised if later on Dr. Rundstrom found cases where even though the sinuses were freely drained there would still be recurrent crusting which would be very hard to ascribe to the sinuses.

Dr. H. L. Pollock said that in 1911 he read a paper before the Academy and gave the various theories of ozena, some of which the essayist did not mention. At that time he also called attention to the fact that the majority of cases occurred in young girls, most of them beginning about the age of puberty. He had one girl between twelve and thirteen years of age under observation, who came to him for ear trouble and he discovered the beginning ozena. At that time he found that there was a horny change in the turbinate which occurred in other diseases, such as osteomalacia and similar changes, and this led them to believe that the whole underlying cause was in some way connected with the endocrin glands. On this assumption he treated a number of cases with the various extracts of the endocrin glands and to his surprise many of them improved greatly for a while and then suddenly reverted back to the state in which he first saw them. This was perhaps because he did not know which of the glands to give and in what dosage.

As to the bacteriologic side, he believed that the fetor of the ozena was probably caused by the Abel's bacillus, but that there was nothing more than the saprophyte there. The secretions are decomposed and we simply find the saprophytic organism, but not the original cause of the disease.

As to the treatment, at the January meeting Dr. Pollock had presented a case in which he demonstrated the method of treatment which had given them the best results. Primarily in every case sinus disease is found; in most cases the ethmoid is involved, but occasionally the frontal and the sphenoid. After cleaning out the sinuses the crust formation still persists, and the only way to obviate this is to fill up the space. The passage of the air dries up the secretion and the crusting and odor persist. This is best overcome by implanting tissue in the septum. They have tried, as Halle did, to remove the lateral wall to the septum, but they found it easier to move the septum out to lateral wall. In many cases they have had excellent results in lessening the discharge. When Hoefer was doing this work Dr. Horn of San Francisco and Murray of Minnesota were given some of the pure culture, and they carried on the experimental work here and tried to produce the disease from this Perez bacillus. Horn claimed to have produced the disease and sent some of his vaccine, which they tried without obtaining any result. Murray reported that he got no results.

They believed the disease was the result of a sinus condition in childhood, rather than a suppurative ethmoid. In their hands the best curative results are obtained by cleaning out the sinus and then filling in the space which the atrophy has caused.

Dr. Robert Sonnenschein called attention to the original article of Perez, formerly of Buenos Ayres but now ambassador to Vienna. In that article he stated that by injecting this coccobacillus fetidus of Perez into the superficial veins of the ears of rabbits atrophy of the turbinate would be produced. These findings were never verified by other investigators.

So far as the word "ozena" is concerned it always has been somewhat confusing. His understanding of the term is that it means a stench, and many cases of atrophic rhinitis do not have this odor. Dr. Rundstrom spoke of the fetid and nonfetid cases of ozena, and Dr. Sonnenschein thought this was a mistake in nomenclature. If ozena means a stench there cannot be a nonfetid ozena.

Dr. H. W. Loeb said that about 4,000 years ago one of the Pharaohs erected a monument to his physician, who cured him of a "stinking disease of the nose," and Dr. Loeb believed that must have been ozena. Since then everyone has been trying to find a cure but has failed. In Bosworth's earliest textbooks it is stated that the cause is a purulent rhinitis. He thought the picture of the mucosa in atrophic rhinitis was so essentially different from that of hypertrophic rhinitis and occurred so much earlier than the hypertrophy, that it was hardly worth while to consider that in connection with the cause and effect.

Dr. Loeb referred to the work done by Dr. Horn, which he thought was very advanced, but without effect. He had succeeded in producing atrophic rhinitis in animals, in which he injected the material obtained from under the mucous membrane of patients suffering with atrophic rhinitis. Dr. Loeb had seen the specimens and thought they looked like atrophic rhinitis. The vaccine treatment was in vogue for a while, but it had done little good.

DR. ALFRED RUNDSTROM (closing the discussion) said that in response to Dr. Morwitz's remark he took a rather skeptical standpoint as to the "wonderful results" of the vaccine mentioned by him. He had both heard and read about those results, but had also heard that that therapy had been given up.

In answer to Dr. Grove, he remarked that, as mentioned, many believed the ozena to be contagious. Dr. Rundstrom, however, did not believe that it was. In instances where he had seen several cases in the same family or many cases in the same locality, he had been able to trace the origin of the ozena to an epidemic of either diphtheria or scarlatina or variola, which had been the cause of the original sinus infection.

Dr. Rundstrom said he wanted to thank Dr. Holinger for his valuable remarks in the discussion. The theory of sinus disease being the cause of ozena was advanced as early as 1866 by O. Weber and has since been formulated in different ways by others. His own work, after he had convinced himself of the correctness of that theory, has been to find the reason that the clinical picture of an ozena was so entirely different from the clinical picture of an ordinary sinusitis. The reason for this difference he believed he had made clear in his paper. Furthermore he had proved that there was a distinct difference pathologically as well as clinically between the fetid and the nonfetid ozena. He had also made the observation which Dr. Holinger mentioned, that one was not able in all cases to show raw bone with the probe; in those cases which he had found after he had cleared the way to the sinus and opened the diseased sinus that the secretion had dried up already in the sinus and formed a crustlike membrane lining in its walls, preventing the probe from reaching the decayed bone. Dr. Holinger's statement that there are cases where even though the sinuses are freely drained there will still be recurrent crusting is undoubtedly true, but the crusting will still be due to the caries of the bone and will not disappear until there is healing of the bone.

Dr. Rundstrom said it was very interesting to hear Dr. Pollock's remark about the osteomalacia. He had also, but only in one case of examinations of many turbinate bones, found an alteration in the bone tissue. He would rather call this alteration a rarefying osteitis. Concerning the method of decreasing the width of the nasal cavity by removing the lateral wall or to move the septum out to the lateral wall, it is the same idea as to increasing the volume of the turbinate bones by injection of paraffin, the danger of which he had mentioned.

Concerning Dr. Sonnenschein's remarks against the name nonfetid ozena, he would not admit that it was a mistake. It was unlogical and he was sorry that it was used in the medical literature, but it was there and it was used in the textbooks. As far as Dr. Rundstrom was concerned, he had a long time ago proposed to cancel the name ozena and substitute it with the more logical sinusitis purulenta foetida et nonfoetida cum rhinitis atrophica.

THE CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday, April 3, 1922.

THE PRESIDENT, DR. ROBERT SONNENSCHEIN, IN THE CHAIR.

Eosin Silver Solution.

DR. ALFRED LEWY presented a 2 per cent solution of eosin and silver, concerning which chemists had reported as follows: "The solution contains no free silver and does not precipitate protein and is not precipitated by sodium chlorid solution. It is not toxic if injected intravenously, and a 5 per cent solution causes only slight irritation."

Dr. Lewy stated that he had tried this preparation out, using it locally in the same manner as other silver salts, and found it was not irritating in a 2 per cent solution. Its great drawback was its staining properties. It must be used in the nose with caution and can be used in the throat if watched carefully. It is slightly analgesic and if used in ordinary sore throat for quite a while pain will diminish. It is apparently useful in superficial infections, but does nothing in deep seated ones. It is not necessary to precede it with cocain. Its principal value to him had been in cases of chronic otitis media where it was possible to get the solution into the tympanic cavity.

Mercurochrome gave Dr. Lewy the idea of trying to get a silver salt compounded with a dye. He now has a chemist trying out copper and zinc both with eosin and other dye stains, in an effort to find one that does not stain as much as eosin. The stain can be temporarily removed with 3 per cent hydrochloric acid, but when water is put on it reappears. He thought the therapeutic value was due to the penetrating quality of the dye; the object of the dye was to carry the silver into the tissues.

SCIENTIFIC PROGRAM.

Dr. Don C. Sutton (by invitation) presented a paper on

"Hyperesthetic Rhinitis of Known Etiology."

(AUTHOR'S ABSTRACT.)

This condition is probably a local manifestation of anaphylaxis to a specific foreign protein.

Dale and others have called attention to the similarity in the reaction of histamin and that of anaphylaxis. But this individual may be desensitized to a specific protein but not to histamin.

The specific anaphylactic hay fevers include the seasonal types, as pollens, trees, grass and compositæ.

Foods: Bacterial and animal emanations of all year type—Animal emanations, foods, bacterial pollens.

Pollen hay fever: 1. Early spring (rose fever), due to trees as willow, poplar and maple, dandelion and occasionally other flowers.

2. Late spring and early summer: timothy, June grass and redtop—daisy.

3. Late summer: ragweed, corn, goldenrod, daisy, sweet clover.

In 59 cases: 55 were caused by ragweed, 1 dandelion, 3 timothy, 1 redtop, 3 corn, 3 goldenrod.

In 73 cases—all causes—55 ragweed, 3 corn, 3 goldenrod, 1 dandelion, 3 timothy, 1 dog hair, 4 goose feathers, 2 chicken feathers, 2 cat hair, 1 redtop, 1 bacteria, 2 cases season hay fever, unknown cause.

Treatment: Twenty-four treated with a pollen extract prepared by the method of I. Chandler Walker: 8 completely relieved, 33 per cent; 10 improved greatly, 42 per cent; 18 relieved or improved, 75 per cent; 6 unimproved, 25 per cent.

Ten cases were treated by a suspension of the ground pollen in olive oil, giving three to four injections at five day intervals just before and during the attack: 4 completely relieved, 40 per cent; 4 relieved, 40 per cent; 2 unimproved, 20 per cent.

Eight cases treated with intravenous peptone solution, as described by Auld, B. M. J.: 5 completely relieved, 60 per cent; 2 improved, 25 per cent; 1 unimproved, 8 per cent.

Vernal conjunctivitis, 3: 1 ragweed, cured with pollen; 2 undetermined; 1 relieved by peptone, 1 unimproved by peptone.

DR. OTTO J. STEIN presented a paper entitled

"Intranasal Injection of Alcohol in the Treatment of Hyperesthetic Rhinitis and Some of the Nasal Neuroses."

*See page 1129.

DISCUSSION.

DR. J. GORDON WILSON said that the interesting papers indicated how very obscure a subject hyperesthetic rhinitis is. Dr. Sutton dealt briefly with some of the modern methods employed in the diagnosis of the numerous factors which produce the symptoms. In spite of the great amount of work done, the primary cause of this susceptibility is unknown.

Hyperesthetic rhinitis, looked at from the standpoint of the rhinologist, may be said to arise from an instability of the nasal mucosa, understanding by instability the loss of power to react within normal limits to factors not unusually present in the air and probably also in the blood. To the question as to what is this instability due, no answer had as yet been given. Dr. Sutton referred to an hereditary sensitivity. There appeared to be no doubt that some families have such an instability, but not necessarily to the same special protein.

When one asks what is actually known about hyperesthetic rhinitis, one soon becomes aware of our deficiency of knowledge. We know that the absorption of vegetable and animal proteins by the nasal mucosa can produce a sensitization, both local and general. Why, then, is it not possible to desensitize by the nose? We know that in sensitized individuals the symptoms of hyperesthetic rhinitis can be produced locally by a suitable protein, and one asks if it is possible to have a nasal mucous membrane sensitized in an individual who is not generally sensitized. A number of nasal membranes which are hypersensitive to pollen are hypersensitive to other things —e. g., adrenalin—so it is possible to "desensitize" by something to which the mucosa is not sensitized—e. g., peptone.

These are a few of the questions which arise to which so far no answer can be given. But in considering the gaps in our knowledge we should also consider the progress that has been made in the last few years. Dr. Wilson has no difficulty in explaining the influenec of the psychic factor mentioned by

Dr. Sutton. To it one applies the principle which Pavloff has demonstrated in his experiments on hypersensitivity from cerebral factors; there is a controlling cerebral influence which may precipitate the symptoms.

Dr. Wilson's treatment is based on the belief that he is dealing with a mucous membrane which is unstable. He finds it advantageous to eliminate factors which tend to produce the symptoms, and so get at least amelioration. As a preliminary he classifies vasomotor rhinitis into two groups, one sensitive to protein reaction, the other to bacterial, this grouping being based on the history and findings in the upper respiratory tract.

Desensitization by proved factors offers the best mode of attack. He removes any cause that appears to increase this instability (polys, sinus disease, etc.), and following this he expects to obtain some benefit. For getting at the fundamental etiology he is relying upon such work as Dr. Sutton and others in this special field are doing.

Dr. Harry Kahn said that for a year or two he had been seeing a number of cases of intumescent rhinitis with general depression and more or less discharge. These patients also showed a definite symptom which had not been referred to in this discussion—namely, diminished blood pressure. He had noticed this hypotension rhinitis in many cases, with the blood pressure around 100. One patient seen recently had a systolic pressure of 90. Most of these cases were in women and were accompanied with more or less general symptoms, pain, hay fever, hyperesthetic rhinitis and rhinorrhea. These cases had yielded to simple treatment, exercise, drinking of coffee, cold baths and abundance of food.

Dr. Edwin McGinnis, referring to heredity in these cases, stated that he had three sets of grandparents, parents and children who were sensitive to ragweed, both small and large. He had tried Dr. Goodale's plan of densensitization, but had decided that he was not going to attempt this any more in hay fever patients, but would use the vaccines and toxins for diagnostic purposes. Before operating upon cases of rhinitis, simple or complicated, they always make the skin tests and in many instances determine not to do any operative work. He cited the case of a child who was sensitive to redtop, timothy and the small and large ragweed. Consequently she had trouble from the last week in May, when the redtop pollenates, until frost. She was very sensitive to the small ragweed and usually got an accompanying infection in the frontal sinus, in the antrum and the ethmoids, which would give her trouble until Christmas time. Attempts to desensitize with a solution of pollen had little effect. He had found that these patients got along very well if they were instructed that they were sensitive to a certain pollen, for they would stay away from it. They had found that injection of hay fever pollen produced difficulty in breathing and started an asthmatic condition. It has been found to irritate the eye when dropped into it, just as it does the nose. In damp weather, when the pollens cannot fly these patients have much less trouble.

Dr. McGinnis thought that the pollen extracts are useful in diagnosis and some people apparently get a certain measure of relief from them.

Dr. Norval Pierce admitted that he had not gone into this subject very deeply, having been appalled by the complexity of it. The terms themselves, sensitization and desensitization, are more or less vague. The fact that there is skin reaction in cases with certain nasal symptoms does not necessarily mean that there is a connection between the two. Several years ago he carried out with the greatest care the instructions that Dunbar gave, but the vast majority of cases were unimproved. He thought perhaps the most fortunate results were gained from the dandelion cases. He thought that the exact cause or causes of these conditions was not known. He cited the case of a young lady with a pronounced vasomotor disturbance of the nose. They were unable to determine that she was sensitive to anything especial, after going through a certain number of foods and of pollens. He then learned that she was being courted and influenced the mother to prevail upon her to desist somewhat. The young man went away and the rhinitis disappeared completely. She had all the symptoms of a pronounced vasomotor disturbance, and Dr. Pierce wondered whether she was sensitive to her own ovarian secretions.

Dr. Pierce thought that it could be stated that it is bad practice in these cases to tear out the architecture of the nose to cure them. That it is absolutely wrong to open up the so-called hyperplastic ethmoids. The one thing that had given him most success in these cases was cauetrization of the four anterior sensitive portions of the nose with trichloracetic acid, meaning the anterior head of the middle turbinates and the adjoining middle meatus, the tuberculum of the septum and the anterior heads of the inferior turbinates. This had given relief for a month or six weeks, when it was repeated.

Dr. Robert Sonnenschein reported the case of a professional gentleman, aged thirty-four, who had had hyperesthetic rhinitis for some years. The principal symptom was that a stream of clear fluid would frequently issue from his nose. Careful examination, including blood pressure, blood counts and urinalysis, was negative, as were radiographs of the sinuses. He was tested with thirty or forty proteins without showing sensitization. Dr. Pierce also examined him carefully, and a small mass, not definitely a polyp, was located high up in the middle meatus of the right side. On his suggestion this was carefully removed, and the anterior ethmoid cells were opened up without disturbing the middle turbinate. This occasioned slight, if any, improvement. Thyroid extract was given for a time. Trichloracetic acid gave considerable temporary relief. Atropin, 1/250 of a grain, three times a day, gave relief without any dryness of the throat or other unpleasant reaction. Real comfort was obtained by taking this for a week or ten days at a time. He has freedom for a short time and then the process repeats itself.

Dr. Sonnenschein confirmed Dr. Pierce's statement that the use of trichloracetic acid gave considerable relief for a time. After the demonstration of a case by Dr. Beck several months ago, at which time the deficiency of certain vitamins was discussed in relation to nose and throat conditions, Dr. Sonnenschein had treated a number of cases with crude cod liver oil, but the results were disappointing. A number of patients examined by Dr. Sutton and found to be sensitized to certain pollens had been relieved.

Dr. Otto J. Stein (closing) said that ordinarily the subject of alcohol excited a great deal of interest and stimulated discussion. He inferred that his method had been used very little by those in the audience, although he recalled several gentlemen in the city who had used it and regretted that they were not present to tell their experiences. Any method of treatment which was valuable in this puzzling condition should be welcomed by everyone.

He had always felt that there were two main factors present in these cases: First, that the patient is hypersensitive or has an idiosyncrasy to some irritant or irritants, and that sensitiveness is due to some condition within the body fluids, be the irritant within the body or without. In the nose the manifestations are principally in the nose, throat and eyes on account of the vast peripheral distribution of the nervous system—that is, the fifth nerve. If irritants are kept away from the susceptible condition there is no manifestation. The great future of the work lies in working out the chemistry of the body fluids, showing why these conditions occur. He believed most cases would be found to be sensitive to more than one irritant and for that reason there are many failures in the vaccine remedies. The element of vasomotor change has been noted in many instances.

Dr. Stein did not go into statistics, feeling that he might be accused of making a statement which he could not substantiate. In the case mentioned by Dr. McGinnis, that continued having hay fever in spite of desensitization treatment, he thought it was possibly another one of the cases in which there was some protein change that was not desensitized.

The statement made by Dr. Pierce, that his best results were obtained by cauterizing the membrane at certain well known areas, was interesting. The patient subjected to irritants at the cauterized areas will not react, because he is desensitized. Dr. Stein thought he would get better results if he desensitized the nasal nerve at its orifice into the nose. Trichloracetic acid he thought was a well known remedy, and Dr. Ingals and others had used the electric cautery. Wonderful results had been reported by irrigating the antrum, and of course some cases are much better after the antrum is washed out.

Another interesting question concerned the role played by the endocrines in changing the body fluids. May not such an imbalance of the various endocrines so affect metabolism as to provoke sensitiveness? He believed this might explain

Dr. Lewy's case and that of Dr. Pierce. He recalled an interesting case of frank rhinorrhea following exposure from a long, dusty automobile ride. It was a vasomotor proposition and the patient was treated by several physicians with all the well known remedies, but was only relieved by Dr. Stein's use of alcohol injections. One injection into the two anterior nasal nerves relieved her. She had no further trouble for some years, when she had a similar attack following another automobile ride. Two injections again produced instantaneous relief from the profuse discharge. Dr. Stein always avoids using suggestion in any sense, because of the peculiar role it plays in the symptomatology.

Dr. G. Thomsen von Colditz presented a thesis entitled

"Treatment of Otitis Media with Tuberculin."

DISCUSSION.

Dr. Harry Kahn asked what dosage was used.

Dr. John A. Cavanaugh asked whether Dr. von Colditz did anything besides give tuberculin.

Dr. Norval H. Pierce thought the paper was very interesting and that a greater number of cases of tuberculosis of the ear is encountered than is realized. The bacillus rarely appears in the discharge, and the diagnosis is difficult. The otoscopic picture or appearance may be misleading. He considered the title of the paper rather misleading and would not expect an ordinary suppurative otitis media that was not tuberculous to be cured by tuberculin, nor would he expect a cholesteatoma to be cured by tuberculin. More attention should be paid to the diagnosis of these cases rather than to inject tuberculin on the barest suspicion. If the discharge is injected into a guinea pig there will be a tuberculous reaction, providing the guinea pig does not die from bacteria which contaminate the discharge. Here, again, diagnosis is difficult. Dr. Pierce believed that in cases with a discharge from the ear that is accompanied by signs of tuberculosis, tuberculin is to be recommended.

Dr. G. W. Boot thought the fact might have been overlooked that a patient with tuberculosis can have a suppurating ear that is not tuberculous, just as there may be a discharging ear in a syphilitic patient that is not syphilitic. In either case, anything directed toward the betterment of the general condition will indirectly improve the suppurative process in the ear. Judging from the description, most of Dr. von Colditz's cases had been largely of that type; not necessarily cases of tuberculosis of the ear, but cases of general tuberculosis with suppurative ears, and improvement in the general health was followed by improvement in the local condition.

DR. OTTO J. STEIN recalled two patients with clinical pulmonary tuberculosis and otorrhea of one ear. They received local treatment at the clinic for some time, with no result except keeping them clean. Both patients went to the Municipal Tuberculosis Sanitarium and underwent treatment for a period of a year and eight months, respectively, and were discharged as cured. The pulmonary condition was markedly improved, but the discharge from the ears persisted. In both cases Dr. Stein did a radical mastoid operation, with excellent results, thus corroborating Dr. Boot's remarks that there may be an otorrhea from a variety of causes not tuberculous in a patient with tuberculosis.

Dr. Stein had had only one experience with tuberculin in the active stage, and it ended disastrously. The patient had a mild tuberculosis and a suppurating ear. He improved for a time under tuberculin, but it evidently was not used correctly, for it lighted up an active process, from which he died. Dr. Stein said that he refused to ever use tuberculin again.

DR. G. THOMSEN VON COLDITZ (closing), answering Dr. Stein, said that he started with the Parke-Davis tablets No. 1, dissolving one tablet in 1 cc. of sterile water. A tenth of 1 cc. is given hypodermically at the first treatment. The dosage is gradually increased until the whole tablet is given at the tenth injection. Then he starts with the No. 2 table, increasing the dosage as before, and so on up to the No. 5 tablet.

Answering Dr. Cavanaugh, he stated that he had varied the treatment in different cases. In some he has given only the tuberculin, besides keeping the auditory canal dry with packing. In other cases he has inflated the ear, using equal parts of iodin, menthol and camphor, and cleansed the auditory canal, in addition to the tuberculin.

Dr. von Colditz considers it very dangerous to give large doses of tuberculin, and gives it in very small doses so as to get either no reaction at all or a minimum amount.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Meeting of April 17, 1922.

THE PRESIDENT, DR. HILLIARD WOOD, IN THE CHAIR.

Tuberculous Laryngitis.

DR. M. M. CULLOM presented Rev. D. D. L., white, aet 38, who first consuted him June 22, 1912, with the following history: Ten years ago a diagnosis of pulmonary tuberculosis was made. Spent two years in the West. Began to get hoarse eight months ago. Had to give up work six months ago. Came on account of great pain and difficulty in swallowing and loss of voice.

General appearance, very thin and frail; exceedingly emaciated; distressing cough; hoarseness; spoke in whispers; temperature 100; weight 103.

Examination of the throat disclosed marked pallor of mucous membrane; larynx presented typical tuberculous ulceration involving epiglottis; arytenoids greatly swollen and ulcerated; typical ulceration in the interarytenod space. Was given for local use in the larynx the following spray: Formalin, carbolic acid, aa, drams 4; sodii biboratis, sodii bicarb., aa, oz. 1; glycerine, oz. 8; aq. dest. q. s., oz. 16; M. et Sig. Dilute with four parts of water and use as a spray six times daily, in DeVilbiss atomizer, turning tip downwards towards larynx.

Patient was advised to live in open air, take milk, eggs and plenty of nourishing food.

Patient was not seen again for four or five years, when he returned, reporting that he had done nothing but follow the directions regarding the spray and method of living. Larynx was badly scarred as result of ulceration, but the active process was entirely arrested, and he was suffering only the effects of traumatism inflicted by ulceration. Had gained a great deal in weight; was apparently in good health.

April 17, 1922. Patient again returned, complaining of hoarseness and loss of voice dating back to attack of influenza first of year. Examination shows remains of old tuberculous laryngitis. Vocal cords red, epiglottis deformed and scarred; no active ulceration. Weight about normal—i. e., 130 pounds. Appearance good; temperature normal. Dr. Cullom does not believe the present hoarseness has any great significance, but is merely a catarrhal laryngitis, which will soon clear up. Advised patient to rest voice, use formalin-carbolic spray and live in open air.

Dr. Cullom presented this case as one of those which have been cured by the use of the above formalin-carbolic spray. He remarked that by all medical theories this patient had no business here, as these cases are generally expected to pass out in a short time.

In connection with this case Dr. Cullom reported also three similar cases in which he had used the above spray, as follows:

1. A woman who had a tuberculous mass in the larynx which shut off all speech; had not talked for nine months; it was difficult to see how she could even breathe. Weighed 80 pounds. Was put on formalin-carbolic spray. Condition cleared up until only a scar could be observed in the larynx, where the tuberculous process had healed. Had slight recurrence past summer upon discontinuing spray. Spray resumed; condition cleared up. Was in office two weeks ago, apparently in good condition. Has regained normal weight—i. e., 100 pounds.

2. A boy upon whom three years ago a diagnosis of pulmonary tuberculosis was made, was sent to Denver, where he remained for three months, when his condition became so alarming that he was brought to his home, and from there to Nashville. Had not slept for three or four nights before reaching Nashville. Presented most distressing picture. Question of tracheotomy was considered; patient referred to Dr. Hilliard Wood for consultation. Had X-ray picture made. Started carbolic-formalin spray. Tracheotomy was delayed for two or three days. Meanwhile it was noticed that breathing was better. Idea of tracheotomy was abandoned. The spray was continued with local treatment of larynx. In a short time the condition seemed to melt away, absolutely disappearing; temperature subsided; patient seemed apparently well and returned to his home and work. Later contracted specific trouble, his Wassermann never falling below four plus. In a

final effort to clear up the specific trouble, Dr. Litterer gave salvarsan, which was promptly fatal, the patient dying six hours later. In Dr. Cullom's opinion, patient would have completely recovered, but for specific trouble, as the larynx was all right.

3. A boy, aet 8. Came in with temperature of 102; typical tuberculous laryngitis; sputum filled with tubercle bacilli; lungs involved. Treatment with spray instituted. Was not seen again by Dr. Cullom, but two years later he was informed the boy had recovered.

DISCUSSION

Dr. E. B. Cayce said that he had used this spray in a number of cases and had found it very valuable.

Dr. Herschel Ezell asked how long it was necessary to use the spray. In what percentage of cases does Dr. Cullom get such wonderful results?

Dr. Leslie Bryan inquired if Dr. Cullom would advise the use of this spray in cases under suspicion, which have not reached the stage of ulceration? If the condition should prove to be nontuberculous, what harm would the formalin do if sprayed into the larynx?

Dr. Cullom, answering Dr. Ezell, said that as he does not do institutional work, his experience has been comparatively limited. It has been his intention to introduce this spray into some institution where a number of cases can be treated and results tabulated. He has used it in quite a number of cases of superficial ulceration about the mouth and larynx, and they have all healed. He has been more interested in the cases of laryngeal tuberculosis, as they have always been regarded as fatal. Of this latter type he has had possibly seven or eight cases in which a cure has been effected. He recalls only one case which went on to a fatal termination.

Replying to Dr. Bryan, Dr. Cullom said that while no great good would probably be accomplished unless ulceration is present, yet he would advise using the spray in cases of suspected tuberculosis, and in the earlier stages of tuberculosis, as no harmful results would follow.

ABSTRACT OF THE SCIENTIFIC PROCEEDINGS OF THE FORTY-FOURTH ANNUAL CONGRESS OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION, HELD AT WASHINGTON, D. C., MAY 1 TO 3, 1922.

Reported by

Emil Mayer, M. D., Abstract Editor, and

C. J. Imperatori, M. D., Associate Abstract Editor.

Harmon Smith, M. D., New York City, in the Chair.

Dr. Harmon Smith of New York welcomed the guests and visualized the present status, scientific, intellectual and social of the association.

The aims and aspirations of this body should not be to fill the full quota of membership, nor to crowd the program with numerous essays, but rather to emphasize merit at the expense of numbers, remembering that "much is not many."

Dr. Smith then made a plea for the amalgamation of otologic and laryngologic associations, for the reason that the coming specialist will be compelled to familiarize himself with the ear as well as with the nose and throat.

Results of the Mosher Operation for Dacryocystitis.

Dr. Charles W. Richardson, Washington, presented the patient. This was an extremely infected case; the infection ran way up to the brow and was tremendously infiltrated. The whole inner side of the conjunctiva was ulcerated over a large area and there was marginal ulcer of the cornea. This was really the worst case I had, and the suppuration has entirely disappeared. This is the most satisfactory type of operation, giving the most perfect results and there is very little trouble with it. Of course, these two were extreme cases. The infiltration in the face was terrific. There would have been a great deal of breaking down of tissues and scarring if it had not bèen operated upon when it was.

Observations on the Art and Technic of Bronschoscopy and Esophagoscopy.

By Thomas Hubbard, M. D.,

Toledo.

Certain precautions are referred to, such as differential diagnosis of the various types of dyspnea, which the bronchoscopist is called upon to differentiate, and in particular that caused by large thymus and peribronchial gland pressure simulating body symptoms.

The function of the anesthetist is mentioned. He is the most important assistant, not only in the matter of posture of the patient, but also to watch for danger symptoms incident to exploration of the bronchi.

Sudden asphyxia is usually caused by shifting of the foreign body to the glottis or into the functioning lung or to the sudden release of pent up secretions below the foreign body, overflowing into the open bronchus.

The cause of the bronchorrhea or flooding below the point of obstruction is due partly to irritation of the mucosa, and in the completely occluded bronchus to the almost continuous high negative pressure.

The surgical lesson is to aspirate deeply the bronchus which has been occluded by the foreign body, to prevent flooding of the open bronchus, and also it is advised to turn the patient onto the affected side to check the overflow.

Syncope during bronchial exploration may be due to vagus pressure or tension due to prying around deep in a bronchus. In other words, faulty adaptation of the axis of the exploring tube to the bronchial axis. Some of these fatalities are probably ascribed to status lymphaticus.

DISCUSSION.

Dr. Chevalier Jackson, Philadelphia, stated that Dr. Hubbard's observations were very interesting, and he could corroborate from his experienec many of the points that he made. He thought, however, that cocain was dangerous in very young children. Dr. Freer used it, he understood, for septum operations in older children; but a large part of foreign body bronchoscopy is done in infants in whom cocain is out of the question. The only objection he had to raise to general anesthesia was in obstructive laryngeal dyspnea, in which the patient was using the accessory muscles of respiration in making a fight for air. If these accessories are put out of commission by artificial sleep the patient is in a dangerous condition. Under general anesthesia, if the breathing is obstructed mechanically by the bronchoscope or esophagoscope, we have again a condition of danger. Personally he preferred to work without anesthesia and with the cooperation of the patient, but each should follow the dictates of his own conscience and experience in the matter. Every laryngologist should be able to examine the larynx without any anesthesia, general or local.

Dr. Hubbard urges strongly deliberate anesthesia. The antonym is precipitate. Dr. Jackson has always opposed precipitate bronchoscopy. Dr. Ingals has pointed out the danger of prolonged bronchoscopies in children. Dr. Jackson's use of the stop watch is to prevent time waste. It does not accrue to the benefit of a child to wait with the bronchoscope in position while a forgotten instrument is hunted up and sterilized. Before we start, everything is supposed to be ready and to go over the operator's ear when asked for.

Dr. Jackson differed with Dr. Hubbard in regard to the toxicity of the peanut. He thought Dr. Hubbard's statement as to flooding indicated a reaction to the presence of a peanut kernel. There is no flooding in a recent case of metallic foreign body. The reaction varies with the individual, just as the reaction to scarlet fever or any other specific infective agent. The adult bronchi do not react to the presence of a peanut kernel.

Dr. Harris P. Mosher, Boston, spoke of two points made by Dr. Hubbard, namely, the need for deliberation in the performance of bronchoscopy, and flooding of the lungs rather than anaphylaxis as a cause of respiratory embarrassment after bronchoscopy. In doing bronchoscopy too much emphasis has been put on time. The stop watch is a dangerous thing in the hands of the beginner. It is all right for a past master like Dr. Jackson, but it gives the rest of us a sense of hurry. In bronchoscopy preparation is just as important as deliberation in its execution. Everything possible should be worked out beforehand. The more bridges that are crossed before the operator gets to them the better. He also believed that flooding of the lungs will be found, as Dr. Hubbard suggests, to play as much a part in the reaction which follows the inhalation of a peanut as the toxicity of the peanut.

Dr. John Murphy, Cincinnati, said he had not seen the hook Dr. Hubbard is using now, but in his early days he had a very unpleasant experience with the hook method. In one case the hook became fastened in the lung, and he spent a few minutes of unpleasant time in extracting the same. Since then he had been very cautious in using the hook, and he had found it not so successful as did Dr. Hubbard. We have all taken most of our lessons from Dr. Jackson, yet the speaker did not use the stop watch, but tries to do the operation with extremely careful preparation that we may be deliberate, yet not lose time before anything is done to the patient.

Dr. D. Bryson Delavan, New York City: The history of bronchoscopy reflects undying credit upon American discovery and invention. Horace Green of New York first discovered the tolerance of the larynx to the presence of a foreign body. Joseph O'Dwyer demonstrated that the larynx would tolerate the presence of a foreign body for an indefinite length of time. Having perfected a tube for the ordinary purposes of intubation, O'Dwyer devised a tube with very thin walls, large interior diameter and straight sides, for the express purpose of introduction into the larynx, in order to aid the expulsion of foreign bodies inhaled into the trachea or bronchi. Many times, at the request of Dr. O'Dwyer, the speaker had experimented with him by means of the ordinary laryngoscope in demonstrating the bifurcation of the trachea through the above mentioned tube inserted in the larynx of a living patient. Dr. O'Dwyer had a clear idea of what he intended to do and wrote a paper on the subject. His studies were interrupted by his death, but not until the practicability of his invention had been proved by actual demonstration. A little later Edison invented the electric lamp, the endoscope was devised, and the combination of that with the O'Dwyer foreign body tube quickly led to the bronchoscope. All who have contributed to the final brilliant result deserve credit, but the real glory belongs to O'Dwyer, the actual originator and demonstrator of the fundamental idea.

DR. HENRY L. SWAIN, New Haven: He would like to say a word in regard to diagnosis. He had occasion in three days to make diagnosis between tumor and foreign body in the larynx, either one of which the medical attendant hoped that I might remove. He discovered paralysis of the posterior cricoarytenoid, which was causing the customary inspiratory and expiratory dyspnea, and the attendant had become confused and thought some foreign body was in the larynx.

DR. HUBBARD (closing): The speaker said that he had neglected to call attention to the one fatal case in the whole series, bronchoscopic and esophagoscopic. Physical examination indicated that the foreign body was probably in the left bronchus. The half kernel of the peanut was readily removed from the right bronchus, but he did not aspirate the left. The family physician reported daily improvement, but when he was called the fourth day the right lung was quite clear, but the left lung was impervious. The baby died soon after. The infant had too much night exposure; he was taken to Toledo Hospital at 4 a. m., and in general there was careless nursing.

Dr. Jackson unduly emphasizes the trauma incident to deliberate operating. It is more important to avoid the trauma of introduction by having a quiet patient. A word as to negative pressure as a factor in flooding of the lung. A plugged bronchus (the accident would naturally occur early in inspiratory effort) would show negative pressure during thoracic expansion until this is satisfied by a fluid transudate and then, as Dr. Jackson says, it would become positive. The fact that fever and all symptoms like pneumonia vanish as soon as the pent up fluid and debris are aspirated indicates that the secretions are the foreign protein which cause fever.

In regard to the hook method of removing safety pins, he used a strong curved hook to turn the safety pin, and the shield hooks are adapted to freeing and shielding the point where it seems advisable to remove the same by forceps.

SYMPOSIUM.

The Radical Frontal Sinus Operation With Report of Cases.

By Fielding O. Lewis, M. D.,
Philadelphia.

In a large percentage of cases in which there is a chronic suppurative process in the frontal sinus, the radical operation affords the best means of a permanent relief from such distressing symptoms as recurring or persistent pain and headaches, purulent nasal and postnasal discharge, alarming vertigo, gastrointestinal lesions and focal infections.

There are a certain group of cases in which some form of radical procedure is unquestionably the only method of treatment, namely, those cases with external fistulæ, intracranial complications, extremely large sinuses, with their often present septal divisions, bone necrosis, severe orbital complications, and when the intranasal operation has been unsuccessful.

By radical operation is meant the Killian or some of its many slight modifications. This cannot be accomplished by chiseling a small hole through the anterior plate any more successfully than a mastoid operation can properly be performed through a small opening in the mastoid cortex.

In a somewhat limited experience in about forty cases operated on radically, a complete removal of mucous membrane, a careful examination of the septa when present, as diseased cells are often concealed at their base, and good nasal drainage are imperative in order to secure a complete cure.

A postoperative deformity depends, of course, upon the size of the sinus and the amount of necrosis of the anterior plate. Those patients, particularly those past the age of vanity, are so delighted with the cessation of the symptoms that they prefer to retain their defect, though sometimes marked, rather than submit to what some have feared might be a return of the old trouble.

The Diagnosis and Treatment of Diseases of the Nasal Accessory Sinuses in Children.

By Cornelius G. Coakley, M. D.,
New York.

The existence of an infection of the paranasal sinuses can not always be readily predicated in the case of a child. The difficulties of examination and the uncertainty of some of the tests are in indirect proportion to the age of the patient. The younger the child the more one has to consider the probability of the infection rather than be able to make a positive diagnosis.

The maxillary sinuses, the frontal sinuses and the ethmoid cells are primarily evaginations of the nasal mucous membrane developed from furrows or ridges in the superior and middle meati. The sphenoid sinuses develop in connection with the cartilaginous nasal capsule, and in a sense are primarily constrictions of the nasal mucosa from the dorsocephalic part of the nasal fossæ.

The speaker divides these into groups according to age.

Group 1. Those between the ages of eight and twelve.

Group 2. Those between the ages of five and eight.

Group 3. Those under five years.

The justification for this classification may be found partly in the stage of development to which the sinuses have arrived at these ages, partly by the certainty with which one may make the diagnosis and partly the treatment required in each group to effect a cure.

The complete examination of a child for suspected paranasal sinusitis often cannot be completed at a single sitting. The nervous child dreads the very sight of instruments and is apt to resent their use. By tact and patience on the part of the examiner and by proceeding with the examination a little at a time we find it possible, at a second or third visit, to examine those in Groups 1 and 2 as satisfactorily as we do our adult cases. In Group 3 we can seldom make what we consider a complete examination, owing to undeveloped nasal cavities.

For a complete examination of the sinuses our procedure is as follows:

A. Anterior rhinoscopy.

B. Posterior rhinoscopy.

C. Anterior rhinoscopy after contracting and anesthetizing the nasal mucosa with a weak solution of cocain, 1 per cent, and adrenalin, 1/20,000.

D. Posterior rhinoscopy, as in C.

E. Transillumination of the frontal and maxillary sinuses.

F. Inspection of each naris in the inferior meatus, middle meatus and the posterior nares with the nasopharyngoscope.

G. Radiographs of the sinuses, anteroposterior and transverse views.

In Group 2 each of these procedures may usually be readily carried out, and from the composite results a very accurate diagnosis can usually be made. Transillumination of the 10-year-old is not apt to be reliable or as valuable as in the 11 and 12-year-old, so much importance is not given to the lack of illumination in the frontal and maxillary regions of the cases of 8, 9 or 10 years. Radiographs of the sinuses in this group are very helpful. The maxillary cavities by this time are sufficiently pneumaticized to show clearly when not diseased. The anteroposterior view of the frontals, however, if apparently cloudy, must in part be judged by their depth. If deep and cloudy they are diseased, but if shallow and cloudy they may not be diseased.

For confirmation of the other evidences of paranasal sinusitis we rely very greatly upon the use of the nasopharyngoscope. With the nasal mucosa anesthetized and contracted with the cocain adrenalin spray there is often too much secretion in the nose for the use of this instrument. The removal of the excess secretion cannot be done by douching or irrigation, as the bubbles of air that are formed in the middle and superior meati and in the nasopharynx prevent one from viewing the orifices of the several paranasal cavities. We suck out all of the visible secretion within the nose and nasopharynx by means of a small metal tube carried into the naris and back to the nasopharynx. The end of the tube is directed to wherever secretion is visible, be it inferior or superior meatus. The pharyngoscope is gently but quickly passed along the floor of the naris until the distal end touches the posterior pharyngeal wall. With lens vertical in this position one may see the anterior surface of the sphenoid, note the degree of inflammation or edema in the region of the ostium sphenoidal and in cases of infection of this sinus usually see a small threadlike white or slightly yellow streak of discharge coming through the ostium. Failing to see such discharge at a single examination does not prove that the sphenoid is not infected, for we have many times not seen a discharge at the first visit, only to find it at a subsequent one.

Group 2. Children Between Five and Eight Years Old.—The same routine of examination is carried out in this group as in Group 1. Posterior rhinoscopy is apt to be difficult or in younger ones impossible. Transillumination is most unreliable in these cases. The frontals scarcely illuminate at all, except in cases of unusual development. Transillumination of the maxillary sinuses is generally poor and unreliable, owing to the fact that the developing and unerupted teeth give an even darkness under the orbit. If one side does show somewhat darker than the other, one never knows whether it is due to an infected antrum or to a lesser degree of pneumatization of the antrum in the stage of development.

Radiographs are of some help, but here, too, great care must be used in reading the plate to differentiate between disease and underdevelopmnt.

The pharyngoscope comes to our aid in finding the secretion at the upper border of the posterior end of the inferior turbinate. The pharyngoscope can rarely be used in the middle meatus in this group owing to the lack of space in the small nares at this age.

Group 3. Children Under Five Years of Age.—In this class we are practically confined to such knowledge of the sinuses as may be gained by radiographs. The reading of some plates of this group by radiographers who have no opportunity of checking up the clinical picture with the appearances of the plate often leads to very wrong interpretation.

Treatment.—In Group 1 the treatment recommended is precisely that which one would use for similar conditions in the adult. If an antrum cannot be induced to drain satisfactorily by keeping the nasal mucosa contracted, a trocar can be introduced under the inferior turbinate and the cavity washed out. We find it usually requires less time and fewer irrigations to accomplish a cure in children than in adults.

In Group 2, owing to the size and position of the antrum and the small frontals, irrigation of these cavities is seldom advisable. Daily contraction of the nasal mucosa with cocain and adrenalin, and irrigation of the nose with a warm saline solution may be relied on to effect a cure.

In Group 3 only irrigations as in Group 2 are necessary.

One of the most important prophylactic measures against recurring paranasal sinusitis is the establishment as far as possible of adequate nasal respiration. The swollen edematous nasal mucous membrane mechanically blocks the outlets for the secretion from the various paranasal cavities. The thorough removal of tonsils and adenoids is obvious. Whether the operation should be performed during an attack or in the interval between attacks must be considered in each case. We prefer, if possible, to operate in the interval, but occasional cases are seen when no progress of the sinus disease can be made until these hindrances to adequate respiration are excised. We find those children with narrow nares and deflected and thickened septa the most difficult to give proper nasal respiration and drainage.

A residence for those who can afford it in a warm, dry climate and a continuous outdoor life may be necessary in some cases to restore the chronically infected mucous membrane lining the paranasal sinuses to normal.

Extradural Abscess Complicating Frontal Sinusitis.

Ross Hall Skillern, M. D., of Philadelphia, reported this case.

A young man of 18 had considerable discharge from the nose, headache and fever following swimming and diving. This was followed by swelling of the right upper eyelid. This swelling was subsequently opened and pus evacuated, leaving two fistulæ. Roentgen examination showed opacity of the right frontal sinus and right ethmoid.

Operation external. Loose sequester of one removed, exposing the sinus cavity, which was filled with pus and granulation tissue. There was a large defect in the posterior sinus wall, exposing the dura. There was a large extradural abscess with two ramifications.

Bacteriologic report of the culture from drain pus showed staphylococcus pyogenes aureus in pure culture.

The patient was discharged four weeks after admission to hospital.

DISCUSSION OF PAPERS BY DR. LEWIS, DR. COAKLEY AND DR. SKILLERN.

Dr. J. H. Bryan, Washington, agreed with Dr. Lewis in everything he said in regard to the need for radical operation in these severe sinus cases. In regard to the Killian method, the speaker was perfectly frank to say that he did not do it. We are operating on Americans, a great many of them are women and they certainly do not want to be disfigured as the Germans disfigure their women. The cosmetic effects are bad in the Killian operation. It is not necessary, except in extensive necrosis, to remove the whole anterior wall. He makes quite a large section of the anterior wall, leaving quite a bridge between the lower end of the resected bone and the ridge. If you take away quite an extensive section and do not remove the entire wall, you will not get the depression. Of course if the wall is necrotic it must be taken away, and there is bound to be a certain amount of depression, but complete removal is not necessary unless necrosis is present. The suture of this wound is important; he coapts the edges carefully and takes ten to fifteen sutures, more if necessary, to bring the edges together perfectly. In many of his cases there is almost an impossibility of determining whether the patient has had an external operation.

In regard to Dr. Skillern's report of a case of extradural abscess following suppurating frontal sinusitis, he called attention to a case of extradural abscess following suppurating frontal sinusitis due to a streptococcus hemolytic infection, reported at the Boston meeting. This patient had undergone several operations before coming under his care, and during one of these operations the posterior wall of the sinus was perforated, and it was through the opening the infection took place, resulting in an extradural abscess. It was difficult to localize the abscess, as it had all the appearances of being in the anterior lobe, but injection of nitrate of thorium through the sinus leading to the abscess demonstrated very clearly that it was extradural. A subdural abscess developed under the corresponding anterior lobe and the patient died of meningitis.

DR. LEWIS A. COFFIN, New York: For the relief of symptoms, yes; for safety of operation as well as further welfare of patients the radical operation as described by Dr. Lewis is often imperatively demanded and offers the only certain relief. I have no sympathy with Dr. Bryan's suggested modification of the Killian operation.

Dr. Francis P. Emerson, Boston, stated that during his last hospital service all children under ten years admitted for a postaural abscess had their accessory sinuses X-rayed as well as the temporal bone. These cases were taken as they applied for treatment, whether they had symptoms referred to the nasopharynx or not. Out of 30 cases so examined 80 per cent showed X-ray pathology in the antrum. In two there was a suppurative ethmoiditis and antrum (maxillary), and in two a pansinusitis on the side of the affected ear.

Dr. James E. Logan, Kansas City, said the character and location of the mucus incrustation in the nose made him believe that the pathology of atrophic rhinitis might find explanation in previously existing sinusitis in childhood. In a paper on atrophic rhinitis previously read before the association he attempted to prove the correctness of this theory, and never had any reason to change his opinion.

Dr. Lee Wallace Dean, Iowa City: The speaker emphasized the importance of nutritional therapy in the management of cases of paranasal sinus disease in infants and young children. Regardless of the apparent condition of the baby or young child, so far as its nutrition is concerned, he found it to be of decided advantage to have not only the pediatrist but an authority on nutrition assist in the treatment of this class of cases. He reported an experiment which had been made at the University of Iowa which has some bearing on this subject. This has given him food for thought. Dr. Daniels, in the metabolic department, fed about twenty-five or thirty white rats, with the expectation of trying to produce by feeding xeroderma pigmentosa. She fed these rats on the following: 18 per cent casein, purified; lard, 10 per cent; salt mixture, 7 per cent; corn starch, 65 per cent. As the result of these feeding experiments typical xeroderma pigmentosa was produced, and Dr. Daniels asked if she might have the services of the chief pathologist to work out the pathologic findings. The rats were sent over to our laboratory, and Dr. Armstrong found that in every rat there was an extensive suppurative pansinusitis. White rats that had been fed on a diet poor in calcium and phosphorus, the rachitic rats, had no sinus disease. He did not report this with the intention of stating that the absence of any vitamin will produce a tendency to paranasal sinus disease, but mentioned it simply because of its interest in connection with nutrition and paranasal sinus disease.

There was a woman in the obstetric service with maxillary sinus infection caused by hemolytic streptococci. She gave birth to an infant who, when ten days old, developed nasal discharge, and the hemolytic streptococcus was present in the discharge. Nasal irrigation, suction, argyrol, etc., were used. The infant developed a suppurative mastoid, was operated upon and the ear became well. At the age of about seven weeks the infant died. It was autopsied by Dr. Armstrong and Dr. Medlar. In the paranasal sinuses much pus was found. They believed that the cause of death in this infant was a suppurative paranasal sinus disease, which persisted in spite of our very best treatment.

DR. OTTO T. FREER, Chicago: The objections to the radical Killian sinus operation are: The danger of permanent diplopia due to detachment of the pulley of the superior oblique muscle of the eye; great deformity in spite of partial removal of the orbital roof, where the sinus is very large; the objection that recovery is obtainable by the less formidable operation of thorough drainage. In an example of permanent diplopia seen by Dr. Freer and following a Killian operation done by another operator the patient found the double vision so distressing that he kept a patch over one eye, ceasing to use it.

While the large intranasal opening that may be made through the nasal wall of the maxillary sinus makes successful treatment of chronic maxillary sinus suppuration easy, the intranasal operation upon the frontal sinus is uncertain as to result, because this sinus is the physical equivalent of an inverted bottle with a narrow neck filled with a thick fluid, pus in the case of the frontal sinus, which fluid is retained in the cavity of the sinus by atmospheric pressure. For this reason even a maximum intranasal opening into the frontal sinus made by enlarging the fronto nasal duct may not give the good drainage needed, especially as the operation is usually followed by prolonged formation of granulations that obstruct the new made passage. This condition is changed at once to the favorable one of the maxillary sinus by through drainage with a rubber drain, size No. 10 to 12, English, the drain being inserted through the brow near, the bottom of the frontal sinus and passed into the naris from above, after the resection of the middle turbinated body, by removal of the anterior ethmoid cells with the curette and by cutting away the internal nasal crest with a burr from above, unless it has already been removal from below intranasally.

Dr. John M. Ingersoll, Cleveland, emphasized the point Dr. Coakley brought out in regard to the interpretation of X-ray pictures by the surgeon instead of by the radiologist. Studying the radiographs of the anterior posterior and lateral views helps very much in determining the condition of the sinuses. The knowledge that we get in that way, plus the knowledge obtained in the clinical examination, is a very material help in these cases. Then, too, the knowledge gained in regard to the size and shape of sinuses helps in determining what should be done in an operative way.

Dr. Vilray P. Blair, St. Louis: We have had thirteen cases of extradural abscess which had no treatment after operation. The first child, five years of age, a simple incision high up in the forehead and removal of sequestrum and inferior drainage relieved the situation. Another case was that of a boy who had pain and swelling above the right eye. He had had radical parasinus operation. In about August the boy came back with an extension of the frontal sinus operation.

The attempt was made to remove all bone from the affected area, using highly concentrated methylene-blue to stain granulations so we could easily distinguish between granulations and dura, which is not always easy to do. Most all of the upper part of his frontal bone for a distance of three inches wide was removed by the speaker and left in one place where this thin layer of fibrin extended out beyond the excision. He had another operation, removed later the bone from this area, where he had left the layer of fibrin, because it had broken down in an abscess. Later he got a blow on the head in this area, and he came back, and then comes the question of removing bone from the curved area of the skull because that is very bad for the patient to remove bone from this area. We watched the case along, making stab drainage here and there, and at this time this heavy fibrin drainage deposit occurred between the scalp and bone, and microscopic examination showed it to be full of lymphocytes, and the whole thing suggested the possibility of syphilitic deposit, repeated Wassermanns having been negative.

Summing up the case: First, as to the history, the streptococcus was found once, and that is perhaps the true cause of it. He has had ten operations, either for drainage or removal of bone, and he has had five blood transfusions, and in the last operation, in spite of preoperative blood transfusion and something to raise blood clotting, the hemorrhage was so full that he had to have packing. The X-ray picture is so typical of syphilis that one cannot help thinking of syphilis, and we know in dealing with the jawbone it does not raise very much disturbance; that sepsis added to it, putting an instrument in, will cause the thing to spread like wildfire.

DR. EMIL MAYER, New York: This splendid demonstration of Dr. Coakley's, showing so clearly the area of accessory sinus disease in children, recalled to his mind the fact that some years ago, when some of us had the temerity to say that accessory sinus disease did occur, it was followed by a learned dissertation by a German colleague to the effect that there was no such thing; that it was an osteomyelitis, and that they were tubercular foci. It has been demonstrated today, what we said rather hesitatingly some years ago, that accessory sinus disease in children does occur, and it is neecssary for us to pay particular attention to the study and care of such.

DR. J. PAYSON CLARK, Boston, asked that in Dr. Lewis' final remarks he state whether the cases have been free of discharge following the operation.

DR. SAMUEL MCCULLAGH, New York: I would call attention to an irrigation apparatus made by Wregg of New York, which practically consists of a Luc ear douche and a small suction apparatus, through which a bottle can be put in the hands of the patient. It has proved the most valuable adjunct in the treatment of sinus disease in children.

DR. CORNELIUS G. COAKLEY, New York: If the Killian operation is not a successful operation it has not been done properly. When it is rightly done, there is no recurrence of infection in that frontal sinus. Since changing the incision the character of scarring has been very much improved. Start the incision in the middle line and carry above and parallel to the external angle. It can be stitched up evenly. The Killian incision always puckers at this side, and this gives a much better result cosmetically. The man who knows how to operate will not have any interference with the superior oblique. If you cut through the muscle you will have interference with the action of the superior oblique, but if you lift up, pull the superior oblique, you go from above or below, and there will not be a particle of diplopia left.

Dr. Norval H. Pierce, Chicago: It has been stated that whenever you perform a Killian perfectly and thoroughly you will never have recurrence. Some months ago he reoperated a case that Killian had operated on eighteen years before, where drainage from the frontal sinus had been entirely obliterated. He cited the case of a young girl, whose condition was alarming from symptoms of meningitis. At the operation he took away the entire anterior wall of a very large frontal sinus, as much as could be retracted. He could not get up to the median and upper portion of the wall, and at this portion there was softening. In order to further explore this thoroughly he would have had to make an incision directly up in the median line and reflect that flap and, as his heart got the better of his scientific judgment, he did not do it. To his great surprise, she became very much better after this operation. Three weeks afterwards the drainage through the nose was perfect, and there was a good big opening, through which the Doctor could get a catheter. The external wound had healed, the temperature was normal, the spinal puncture showed normal fluid, the pulse was normal, the patient had gained in weight somewhat. The only thing that aroused any suspicion of an internal trouble was that the disposition of the patient had changed. She was irritable, cross; she was getting up at nights apparently without any reason, and trying to wander out of the room. Following an automobile ride she vomited, had headache, the temperature rose; spinal puncture normal. She was put on the operating table, and we abandoned our esthetic sense and made a free incision over the dural portion of this wound and the eyebrow and turned back the flap. We had not turned back the flap until out gushed the pus. Then we found there had been a perforation of the bone. This was enlarged down into the frontal sinus, the posterior wall of the sinus removed and about one and a half inches downward and outward was a fistula through the dura which led into a brain abscess that contained at the time two ounces of pus that we could collect by sponging. This is the abscess of a silent area of the brain, with normal temperature, with normal cerebrospinal fluid, with normal pulse and, in fact, with no symptoms other than a slight accentuation of the normal mental peculiarities of the patient. The patient recovered.

Dr. Lewis (closing) : If he sinus is large, and has accessory cavities, one must remove every vestige of disease to cure the patient. This can be accomplished through a small external opening, except where the sinus is extremely small. There has been no permanent diplopia in any of the cases. There have been transitory diplopia in a few of the cases.

Answering Dr. Clark: As far as he knew, the cases have been free of discharge following the operation. Relative to the incision described by Dr. Coakley, he stated that he has tried it in a few cases, but felt that it did not give sufficient room to reflect the whole orbital wall, which is so often essential.

Dr. Coakley: There was no odor to the discharge, but believe there might have been. As far as nutrition is concerned, practically all the cases are under expert care of pediatrists of the city.

Shawl Pin Extractor.

Dr. Chevalier Jackson, Philadelphia: Dr. Tucker devised this forceps that will not only prevent losing the pin, but bring it back parallel to the axis of the bronchoscope. The least little touch will bring it in. It throws the long axis of the pin parallel to the long axis of the bronchoscope. Simply press it and it comes right in. The nearer the point you get in general the easier your troubles will be, but the main consideration is when the point is stuck in and you get the point out with the ordinary forceps, the pin gets crosswise the end of the bronchoscope. This forceps throws it back parallel. All you need to do is push it down. It is a most ingenious thing.

Incisor for Retropharyngeal Abscess.

DR. REUBEN SMITH: It is just a simple instrument for opening retropharyngeal abscesses. The blade is so placed that it faces the posterior wall and by depressing the handle it incises the abscess.

Modern Commentaries on Galen—The Organs of Respiration and the Voice and their Physiology.

BY JONATHAN WRIGHT, M. D.,

PLEASANTVILLE, N. Y.

In his introduction he shows that the majority of physiologists of antiquity formed an idea of what the anatomic structure should be to account for or serve for some physiologic function, and despite the continued obtrusion of anatomic facts they ignored them or actually distorted them in the interest of their theories.

Voice production, according to Galen, was caused by vibration of the tracheal cartilages by its lengthening and shortening and by its dilatation and contraction.

"Not simply in expiration is the voice formed, but yet the blowing of air is the real substance of the voice." The immovable state of the tracheal cartilages had to do with voice production and it was their motion that has to do with respiration.

In the days of the ancients the importance given to the length of the trachea pervaded the lay mind, and we find it formulated in the resonant lines of their poets.

When an outsider, Garcia, showed us the workings of the vocal cords, we fell into line with a sentiment which overshot the mark. Galen no more forgets the moving and movable laryngeal parts than we entirely neglect the resonant parts of the organ of voice which lie below the glottis.

SYMPOSIUM.

Vasomotor Disturbances of the Upper Air Passages and Sinus Disease.

DR. J. L. GOODALE, Boston: Conclusions: A group of vasomotor disturbances characterized by vasomotor rhinitis or by asthma exists, standing in relation to chronic infection of the nasal sinuses. In vasomotor rhinitis the sinusitis is probably the direct cause of the disturbance. In asthma of this type it is not wholly clear what part of the production of the disturbance is played directly by the absorption of bacterial proteids from the sinuses. There is reason to believe that the sinusitis has an etiologic relationship with the associated bronchitis. Relief of the sinusitis is usually followed by diminution in the bronchitis and asthma, unless the bronchial lesions are old and extensive or associated with myocarditis. Operative treatment of the sinusitis consists of adequate removal of diseased conditions in the ethmoid bone with the establishment of large openings into the antra to permit the spontaneous evacuation of the antral fluid.

Observations in Hay Fever.

By I. Chandler Walker, M. D.,
Boston.

Hay fever is divided into two groups, seasonal and perennial. The perennial variety is often caused by inhalation of the proteins of the hair, dandruff or skin dust of the horse, dog or cat and other fur bearing animals.

The inhalations of plant pollens may predispose to both varieties of hay fever caused by pollens, such as timothy, grass and ragweed. Inhalations of the flour of cereal grains, face powders and dust in various occupations are causal factors.

Another group of causes lies in the ingestion of food proteins. Among the common foods that seem essential to test routinely are eggs, wheat, milk, rice, corn, oats, potatoes, beef, lamb, pork and chicken.

The above mentioned two types may be determined by means of skin or cutaneous tests, the technic of which the author describes. The treatment consists of the elimination of the offending substance.

A third type is due to bacteria, and these are treated with vaccines, chiefly of the autogenous variety. A fourth type is due to pathologic conditions in the nose.

The causative agent of the seasonal variety is usually the pollen of dwarf ragweed in the East and the giant ragweed in the West, and sometimes it may be caused by the pollen of trees. The summer type is occasioned by the pollens of lilies, sorral, buttercup and grasses. The rose is rarely a cause.

Later types are limited to the pollens of the grass family. The autumnal types are due to the goldenrod, sunflower and ragweed, the latter being the principal causative agent. It is essential to do cutaneous tests with various dilutions of the pollens or pollen protein before giving treatment.

The method of treating preseasonally with pollen extracts is fully outlined and the author's results given. He finds that the August and September variety is due to the ragweed pollen, and he gets his best results from the treatment of the ragweed preparation. Preceding and during the season treatment with pollens is described.

Seasonal hay fever due primarily to foods has not come to his notice. It may be occasioned by animal emanations and hay fever.

Satisfactory preseasonal pollen treatment yields excellent results. When this treatment fails, benefit may result during the season from an autogenous nasal secretion vaccine.

The General Treatment of the Asthmatic.

By Francis M. Rackemann, M. D.,
Boston.

There are many causes of asthma. These cases may be grouped under five main headings, which with the percentage number of cases in each group are as follows:

1. Pollen asthma	23.1%	
2. Dust, including horse and other animal emanations	11.7%	
3. Food asthma	2.9%	
Total extrinsic asthma		37.7%
4. Bacterial asthma of various types and perennial asthma	24. %	
5. Reflex asthma	12. %	
(Emphysema, primary and secondary)	17.2%	
Unclassified	9. %	
		62.2%

The type of asthma tends to change—in that simple, uncomplicated types, perhaps due to protein susceptibility, often become complicated by bacterial infections and thus lead ultimately to secondary perennial asthma and secondary emphysema. The possibility of such a change should be recognized in order not to overlook the underlying primary factor.

In the study of each patient, the general condition is often of prime importance, especially in the reflex cases. A thorough physical examination is always essential, and an examination of the nose and throat by a specialist should be part of the routine, not only because an important source of trouble may be discovered, but also because the character of the mucous membrane may suggest the nature of the cause of the asthma.

As a result of the original study, less than 10 per cent of the cases were advised to have some local operation on the nose or sinuses. However, such an operation caused a definite improvement in 60 per cent of these patients, and in all but one it was the only treatment given.

On the other hand, local operations had been performed on approximately 25 per cent of the cases before they came under observation and without improvement.

These facts would urge that the results of the treatment of asthma will be greater, according as the study of the patient as a whole has been more thorough and complete.

Dr. Emil Mayer, New York: The speaker asked Dr. Walker if the solutions of these pollens keep indefinitely? In other words, can he use a solution of a pollen made this year for next year?

Dr. Henry L. Swain, New Haven: It may be of some interest to Dr. Rackemann to learn that some twenty-five or thirty years ago the speaker wrote a paper entitled "Asthma, Its Intranasal Origin and Surgical Treatment," and at that time grouped together a lot of cases and stated that about 70 per cent of all cases will be benefited by treatment. The observation at that time seemed convincing that if we only gave enough attention to asthma in the intranasal way that we could cure all of them.

He cited an instance of a recent patient who had asthma only coming on when he danced with blondes. He could dance with any old brunette with impunity. Finally, it was found that it was due to a face powder used by blondes, which settled the whole situation.

DR. JOHN E. MACKENTY, New York: There is no question that operation first and vaccines afterwards give a much better result. He had three cases this winter with apparent cure by removal of the tonsils alone, one quite remarkable case, who had severe edema; the tonsils were removed; has never had an attack since.

DR. MAX A. GOLDSTEIN, St. Louis: The speaker asked for how long the immunity exists? Whether that means a recurrence of the desensitization, or whether in their experience they can gauge the time for which immunity may go on.

DR. GORDON BERRY, Worcester, Mass.: The speaker asked Dr. Walker whether in his research he had found that foods which the patient had been forced to eat might be foods that later proved poisonous.

DR. WILLIAM B. CHAMBERLIN, Cleveland, asked what the experience has been from the stock preparations from commercial concerns.

DR. GREENFIELD SLUDER, St. Louis: Asthma as a nasal reflex is the chief thing that has interested him. He sends asthmatics to the internist to eliminate the other possible causes before the nasal treatment is taken up, which is somewhat at variance with Dr. Rackemann's observation that they had all been operated upon the nose first. Asthma as a nasal reflex seemed to be almost uniformly either from the nasal (sphenopalatine or Meckel's) ganglion or the sphenoid. Much has been said today about asthma, but there is no explanation of how the asthma arises. In a paper read by the speaker he gave forth some speculation on the production of asthma as a nasal reflex and cited some cases. There was one in whom asthma could be produced by mechanical or chemical irritation of the nasal ganglion. He repeatedly produced asthma by the application of silver nitrate solution over the sphenopalatine foramen, which is uniform to the ganglion. He injected the ganglion and for some five years now she has been free of asthma. It is a matter of speculation as to how nasal asthma may occur in some persons and not in others with the same exciting cause. If you imagine a reflex starting in the nasal ganglion and the anomalous arrangement of the fibers, which does not seem to be an impossibility in speculation, inasmuch as the anatomy varies very much, to have the fibers of the nasal ganglion, instead of being relayed in the upper cervical, to be relayed in the lower cervical. That is in absolute contradiction to the statements of the great workers with the sympathetic, Langley, Gaskill and Anderson, that the sympathetic system is the efferent system only. It has seemed to him for a long while that there is a variety of toxins produced from a variety of organisms and that they do not always form the same clinical phenomena, as, for instance, the cold in the nose may have been the particular thing that exploded the asthma. In the course of Dr. Walker's paper the question arose, why did some of the patients have hay fever only and why did some add asthma to the symptom complex? He believed that the sensitiveness of the sympathetic system varies. Possibly it is explained by just what he said concerning this one particular coryza in this patient; possibly the sympathetic was more sympathetic than at some other time, but be that as it may, the variation is well marked, shown also in this particular type of cases. Sometimes they will explode with the asthma without anything else and other times with perfect classical lower half headache. He heard of a case where a patient had an inguinal hernia and asthma and the inguinal hernia, for some reason, had been delayed and was finally operated and the asthma was cured. Later on an inguinal hernia developed on the opposite side and the asthma reappeared.

Dr. D. Bryson Delevan, New York, said that this excellent symposium recalled the pioneer work done by Daly, Roe, John Mackenzie and other members of this association, and stated that another milestone in the progress of our knowledge of hay fever had now been passed.

The first instance he himself had observed of coryza being caused by sachet powder was in the case seen many years ago of a vigorous infant of six weeks in whom exaggerated symptoms of hay fever were caused by violet powder used to perfume his layette. Upon the removal of the powder, the coryza quickly vanished. Soon after this observation, a lady consulted the speaker, giving a similar history, and with the discontinuance of the violet powder, a like successful result was obtained.

Dr. Paul Carton, attending physician at the Hospital at Brevannes, near Paris, France, has recently published an elaborate treatise upon this and kindred topics, entitled: "Traite de medecine, d'alimentation et d'hygiene naturistes." This work contains some interesting ideas in connection with the study and treatment of hay fever.

Dr. Joseph L. Goodale, Boston, asked Dr. Walker if he has made any observations to determine whether disturbances caused from willow leaves are anaphylactic in character, or due to the mechanical irritation from the leaf hairs.

In regard to the collection of pollens, we must remember the possibility of contamination. For instance, if one gathers pollen of the plant surrounded by grass, grass pollen may adhere to the anthers of the plantain, and our inferences as to sensitization to pollen may consequently be erroneous.

Dr. Walker (closing): With reference to the subject of the pollen solutions, the first question asked, we find that the solutions at times deteriorate each year. The greater the dilution the more the deterioration. It is quite possible to keep a concentrated solution like 1 to 100 perhaps two years and get fair results, but we don't do that. We keep our pollen as gathered in a powder that will keep indefinitely. From that powdered pollen we make extracts each year, thus avoiding holding them over. The gentleman asked about a dislike for common food. That is his greatest help in asthma. He tries to get some history from the mother that the child disliked the food and would almost say nine times out of ten the food that the child disliked and was forced to eat was the cause of the trouble. Often the child is punished when he first refuses to take eggs. The child refuses them at first through instinct. The mother recognizes later, on recalling these matters, that the child did not like eggs; through repeated punishment the child is forced to eat eggs. Sooner or later it is evident that eggs do upset the child. It may be by angioneurotic edema, vasomotor disturbance, true attack of asthma, or vasomoter upset, but it is frequent that a child dislikes food and very often the food it dislikes causes symptoms. The patient may like carrots and eat them every day; in fact, he had a patient who ate carrots, was so fond of them, and they were evidently the cause of asthma in her case.

In reply to Dr. Goodale he would say that it was the protein in the hairs on those leaves that caused trouble, since macerating these hairs and extracting what we call protein from them, we call it protein because it gives protein tests. It may be a toxin as well and giving extracts in dilution protects and in this extract there is no hair. Therefore, the treatment does not consist of anything that could mechanically irritate, although the mechanical irritation may help sensitization; that is, the bristles and spurs may help these things to stick in the mucous membrane,. It is really the protein in the hairs and in the food, an animal emanation, that is the cause. He would rather prefer to say idiosyncrasy than sensitization, but as anaphylaxis fits in with the theory; until we know some better word we had better stick to what has been shown us in the past.

In regard to the stock pollen extracts: In past years results were not very good because they grouped the pollen. They were treating autumnal hay fever with a solution of ragweed, sunflower, goldenrod and even daisy, which pollenates in July. They were making polyvalent vaccine, whereas, what the patient needed was ragweed, and was getting three or four things which was keeping down the dose of the thing they needed. Later, through the efforts of the Government, they are putting out extracts of the proper strength.

DR. RACKEMANN (closing): The speaker expressed his appreciation to Dr. Sluder for his remarks. We are tempted to think only of the exciting causes of asthma and to overlook the fact that there is a fundamental basic cause of asthma about which we know almost nothing. This basis should apply to hay fever, urticaria, etc., which are probably in the same biologic group. In other words, there must be some method of explaining why certain individuals have hay fever and other people do not. Various thoughts occur in this connection, for example, one thinks of some endocrine gland disturbance, but when we see the various types of people who have hay fever and when we see the myxedema and goiter patients with asthma such a thought is almost absurd.

Another theory is that asthma depends upon some abnormal tone through the great vagus or through the sympathetic system; but recently Mackenzie of New York has tested asthmatics with repeated injections of pilocarpin, on the one hand, and with adrenalin on the other, and finds that approximately equal numbers of asthmatics react to each drug without any constancy.

Finally, we must remember that asthma is a symptom; that it occurs in cardiac disease and may even be due to a foreign body in the larynx. He recalled one patient with a tumor of the larynx which was found to be a gumma; antisyphilitic treatment cured the tumor and cured the asthma. Dr. Sluder's theory is very attractive and by it we can explain a large majority of the cases, he believed. As regards the hernia operation, he called attention to the fact that ether anesthesia given to almost any case will result in definite relief of the symptoms, for some time, but this relief is only temporary. Furthermore, infectious diseases of definite severity often bring about a relief of asthma for a time. This seems to be true mostly of respiratory infection either of the head or the chest. It would fit in with Dr. Sluder's reflex theory if we could show that not only do foreign proteins inhaled through the nose act on the nasal ganglion, but that ether anesthesia and respiratory infections produce their effects in the same manner. He was very glad indeed to have heard Dr. Sluder.

Infections Following Tonsillectomy Under Local Anesthesia.

By George B. Wood, M. D.,

Philadelphia.

One of the surprising features of tonsillectomy is that in spite of the impossibility of making and keeping the operative area sterile, there are few serious cases of sepsis following it, even though every tonsillectomy wound does show some infection in that there is a bacterial invasion of a certain amount of tissue with an inflammatory reaction greater than is necessary for the mere healing of the wound. That serious infections are not more common is due to the open nature of the wound affording free drainage, to the mechanical action of the saliva in washing away the bacteria and probably also to its germicidal action. Cases of serious septic conditions following tonsillotomy and tonsillectomy under ether have been reported, but it has been the speaker's fortune to have been spared the tragedy of a septic fatality from tonsillectomy, nor does he personally know of a fatal case of local sepsis when the operation was done under a general anesthesia. Up to last fall he could have said the same thing also in regards to tonsillectomy under local anesthesia.

The writer then detailed a series of cases of infection of the tissues of the neck following tonsil operations.

The regionary infection which may follow from infiltration of the tissues in the region of tonsil can be of three types:

First, the infection may begin in the tissues internal to the superior constrictor muscle, including the palate and the faucial pillars. This would lead to a condition resembling peritonsillar suppuration, and though productive of considerable discomfort, is not apt to be followed by a fatality.

Second, if the needle penetrates through the superior constrictor muscle, which can be easily done if the injection is made deeply through the anterior pillar of the fauces, the infection then starts in the pharyngomaxillary fossa, the importance of which fossa as a serious location for suppurative processes has been so well shown by Mosher (The Laryngoscope, June, 1920). To refresh our memories, the speaker presented the anatomic relation of the tonsils. Abscesses starting in this fossa tend to perforate the lateral wall of the pharynx, but may follow the carotid sheath downward and open into the larynx, esophagus, or possibly the pleura.

Third, an infection of the first or second types usually produces an adenitis of the deep nodes of the neck which are situated on the external surface of the carotid sheath.

The removal of tonsils under local anesthesia is a well established and justifiable surgical procedure, but that it is absolutely free from danger, as has been claimed by some of its most enthusiastic supporters, is to my mind an unwarrantable conclusion. The real danger, as far as sepsis is concerned, is in the penetration of the injecting needle through the superior constrictor muscle.

Some of the Local Unpleasant End Results in Well Performed Tonsillectomies.

By Charles W. Richardson, M. D.,
Washington.

The writer called attention to the tendency of tissue to replace or attempt to replace tissue lost through operative procedures or accident. This is noted in lymphatic tissue —more active the earlier in life that it is subject to operative intervention.

The first group will include those efforts of nature to replace lymphatic tissue removed from the tonsillar fossæ by hypertrophy glandular tissue in the immediate neighborhood.

A. Hypertrophy of glandular tissue in the lateral pharyngeal wall, posterior to the palatopharyngeal fold.

B. Hypertrophy of the lateral borders of the lingual tonsils.

C. Development or hypertrophy or glandular tissue in the palatopharyngeal fold.

The second group is composed of those cases that develop unduly granulation tissues in deep fossae which does not undergo absorption but organizes with the appearance here and there throughout its structure of lymphatic tissue.

Allied to the above mentioned is the redevelopment of island or lymphatic tissue to large masses of lymphatic tissue in the tonsillar fossae.

Adhesion or shortening of the anterior pillar by its adherence to the dorsum of the tongue, and the adhesion of the lower lateral wall of the fossae to the side of the tongue form another group.

DR. JOHN F. BARNHILL, Indianapolis, said that he would report a case somewhat similar. A woman 32 years of age, a professor in one of the eastern colleges, came to his office, had the usual preparation, usual tests such as we make before tonsillectomies, had a local anaesthesia by novocain, almost a bloodless dissection operation, no subsequent disturbance whatever in so far as the tonsillectomy was concerned, but two days later she had a severe chill, with high temperature, a septic condition, and died of lung trouble later on.

Dr. Richardson reported what we all see, namely, that the best tonsillectomies do not always yield the best results and it seemed to him that those that seemed the most complete sometimes yield the worst results, because there sometimes occurs subsequently in these very complete cases an accumulation of new lymphoid tissues behind the pillar. Anyone who dissects these new masses out will find they are much larger than they appear on the surface, since when they are dissected out there is a very deep fossa left. Dr. Richardson omitted to mention one class of case in which there is no return whatever of any tonsillar or lymphoid tissue, but in which the patient makes severe complaint of dryness or other annoying discomfort in the throat. Sometimes the complaint follows what, judging by every test, would be called a perfect tonsillectomy, is that taste is impaired or perverted. It is not easy to account for such complaints, and all treatment seems useless. They wander from one specialist to another and some become pronounced neurotics. Injury to branches of the hypoglossal nerve are usually assigned as the most probable cause of the complaint.

DR. HANAU W. LOEB, St. Louis, said that both these papers are exceedingly timely and later he thought that there were questions to be determined by the Committee on Tonsil Investigation.

He knew of a number of cases that he had been able to collect of hitherto unpublished deaths from local infections and general infections from tonsil removals, both under local and general anesthesia.

He said that in a record of 332 cases hitherto unpublished, of deaths from minor operations he had succeeded in tracing a number of cases of a little different type from those mentioned by Dr. Wood. There were cases of intense edema coming on very soon after operation with death resulting in a very short time. There were some cases of general infection, also.

Under improved method of local anesthesia he found that the Sluder operation was really beautiful and we so far have had no bad results. In reference to the paper of Dr. Richardson, he thought that also was timely. He too, had seen the cases like those he mentions. namely, of increase in the lymphoid tissue just posterior to the anterior pillar of the palate so large they had to be snared off. In a year or two some go down. In others there is recurring acute inflammation which attacks the lateral wall of the pharynx.

Dr. Lewis A. Coffin, New York, said that he preferred local anesthesia where he felt his patient could stand it. Children under fifteen years of age should never be subjected to a tonsillectomy under local, and only such adults as are of such mental stability and temperament as to stand it without much shock.

Most of us have had such experiences as Dr. Richardson has described and one of the best points of his paper is his recommendation of honest charity, and honestly telling the complaining patients that the Doctor did good work and that they should go back to him.

Dr. John M. Ingersoll, Cleveland, said that Dr. Wood's interesting paper brings out a point which should be emphasized. In spite of every care these infections do occur. Many men doing tonsillectomies know that they are operating in an infected field and for that reason they are less careful of their technic than in doing mastoid operations. Extreme care in the technic of tonsil operations may eliminate some of these local infections and at least gives one the feeling that the infection happened in spite of every precaution.

Dr. Elmer L. Kenyon, Chicago, said that he was wondering what a professional singer might think if she heard this discussion.

Good singing requires excellent control of the palate. One of the results not uncommon, indeed seriously common, of excellently performed tonsillectomies was the destruction of the action of the palatoglossus muscle.

In any well performed tonsillectomy hemorrhage may occur. Hemorrhage may lead to the necessity of working upon the very muscles that are so precious to the singer. The result may be infection, injury, adhesions; the final result may be decreased delicacy in these parts for the purpose of singing. The thing to overcome with the singer if we do him good by performing tonsillectomy is his state of mind. It is that conquering of his psychology that has got to come before he can be treated by laryngologists as he should be. He is in a state of fear for his voice. Somehow or other we have got to get at the singer from a different way than we go at the ordinary individual in these tonsillectomies.

He wished to put forward this idea, namely, the idea of performing on the singer an intracapsular lymphoidectomy. If you leave the capsule in you do not have to get in contact with the muscles so precious to him.

Dr. Max A. Goldstein, St. Louis, said that he addressed his remarks solely to Dr. Richardson's paper. We are criticised very often by the general profession and public because we do not remove from the side of the throat every bit of tissue that is there. If we would educate the public and general practitioner to the fact that we attack only one group of lymphoid tissue and that it is impossible to remove all the lymphoid tissue on that side of the throat and that it is quite possible that having removed this mass there may develop in that area a small nodule which in time may grow to look like a tonsil. Just as soon as we educate the public and general profession to this, just so soon will distressing criticism be obviated from our practice.

Dr. Greenfield Sluder, St. Louis, said that lymphoid tissue after removal of the tonsil took on frequently a compensatory action. He defined a perfect tonsillectomy and said it is not the appearance of the fossa after removal that was important, but the examination of the specimen where the capsule was absolutely intact. The lingual tonsil may project itself perhaps upward and behave as the original tonsil.

The case he had particularly in mind was a perfect tonsillectomy in the year 1908. For ten years that boy, who was twelve years old at the time—for ten years that result stood an absolutely perfect result. On one occasion he looked again at this perfect result and to his amazement in the absolute center of the fossa, and not lateral encroachment, presented a lymphoid follicle probably three-sixteenths of an inch in diameter. Now after ten years of such perfect standing result to find a lymphoid follicle develop in the scar base of the fossa was a matter worthy of record and appeals again to the charity Dr. Richardson recommends in the criticism of our confreres' results.

Dr. George B. Wood, Philadelphia, said that he had seen cases similar to those reported by Dr. Sluder: Namely, the recurrence of lymph follicles on the smooth scar of a tonsillectomy wound several months after the operation had been done. He also called attention to the possibility of pockets forming in the lower part of the tonsillar fossae following a complete tonsillectomy where the lower part of the plicatriangularis had not been completely removed. This is due to the adhesion of the edge of the plica to the lower portion of the tonsillar wound.

Dr. Richardson (closing) said that he had seen excessive dryness of the pharynx and that it was one of the most annoying symptoms patients complained of. He thought that they were largely due to injuries of the nerves. He said that he was asked to define a perfect tonsillectomy. Dr. Sluder gave his definition; a tonsil removed within the capsule.

On the Treatment and Prognosis of Carcinoma of the Larynx.

By Professor Waichiro Okada,
Tokyo, Japan.

During more than twenty years of academic and practical activity, the speaker employed various methods in operating on 141 cases of laryngeal carcinoma, and the results obtained and the facts observed have led him to views differing somewhat from those of the various authors. First of all, he spoke of the surgical methods he used and the results obtained, following up with a comparative criticism of other methods taken from the literature on the subject, and ending with some general conclusions on the subject of laryngeal carcinoma.

The methods employed were five in number, viz.: (1) Endolaryngeal excision; (2) thyrotomy and excision of soft parts; (3) partial extirpation; (4) semilateral extirpation, and (5) total extirpation of the larynx.

THE ENDOLARYNGEAL OPERATION.

This method was first employed by B. Frankel with good results on laryngeal cencer, but on account of uncertain results was rejected by most surgeons.

With his endolaryngeal operations he has been well satisfied, but unfortunately in the majority of cases of laryngeal carcinoma the operation cannot be resorted to because when the patient come to us it is already too late. He emphasized that the indication for this minor operation must strictly be that the tumor is sharply defined in a small place. In every case of such nature he is not satisfied with only the ordinary laryngoscopic examination, but tries to examine the ventricular space by the laryngoscopia ventricularis, in order to see if the ventricular arch is already affected or not, and examine besides any possible affection of motility of the vocal ligament with the aid of the mirror. These two points he considers of the utmost importance to ensure the possibility of a successful endolaryngeal operation.

THYROTOMY.

This method is said to have been first used by Solis Cohen in 1868 for laryngeal carcinoma, but has come into vogue only later on the recommendations of Felix Simon, Otto Chiari, Hansberg, Jackson, etc., against early forms of laryngeal cancer. Felix Semon, indeed, prefers this thyrotomy with the excision of the soft parts to the endolaryngeal operation recommended by Frankel under similar indications, as being not dangerous and prognostically much better and for the preservation of the voice much more favorable. He was also of the opinion that the estimation of the extent of the infiltration could not always be properly ascertained by laryngoscopy, and the excision of the tumor by the endolaryngeal operation is not always sufficient. But the speaker maintains that a smaller operation should if possible be resorted to, if it can be presumed that all diseased parts can properly be removed. Thus he had little opportunity to use thyrotomy, because he employed the former method where other authors resorted to thyrotomy.

PARTIAL RESECTION AND (4) SEMILATERAL EXTIRPATION OF LARYNX.

Both methods were formerly frequently resorted to in the treatment of laryngeal cancer, seldom with good results. He has therefore always been of the opinion that sharply defined semilateral or localized cancers, as such, of the epiglottis, which had penetrated rather far, but which appeared as if easily removable by semilaryngeal or partial resection of the larynx, should, from the point of prognosis, at once be totally extirpated.

TOTAL EXTIRPATION OF THE LARYNX.

Notwithstanding the good results of thyrotomy and laryngofissure obtained by such renowned specialists as Felix Semon, O. Chiari, Ch. Jackson and others, and of the endolaryngeal operation by Frankel and himself, they can only be used in a limited number of cases, as cancer of the larynx usually comes before the specialist in already relatively advanced stages. Therefore, the principal operation will always remain the total extirpation of the larynx, as only in this way can all pathologic processes in the larynx be radically removed, and by further improvement of this method the inoperable cases may in future be more and more lessened in number.

In 1881, at the surgical congress in Berlin, Gluck published his experimental treatise in which he showed that in extirpating the larynx, or more generally expressed in all operations, which so often cause death by pneumonia through the entering of foreign bodies into the air passages, an absolute guaranty against this form of pneumonia could be established by prophylactic resection of the trachea and circular sewing up of the central stump of the trachea into the epidermis.

In the years following he practiced this method with excellent results on many patients, and thus Gluck's method of total extirpation of the larynx has been brought to its present standard of excellence. The principal procedures of it are: (1) Complete exposure of the larynx before opening the air passage. (2) Severing the larynx from the pharynx. (3) Immediate closing of the pharynx defect while the larynx is drawn far out of the wound. (4) Severing the larynx from the trachea. (5) Circular sewing in of the lower stump of the trachea into the skin wound, and (6) closing of the whole wound by sutures.

GENERAL REMARKS AND CONCLUSIONS.

From the facts presented here we can clearly see that we are in the period of achievement as regards the treatment of laryngeal cancer and that Virchow's words have been realized: "If cancer is, in its beginning and for some time longer, a local affection, it must be possible to cure it locally at this stage." And there is no doubt that the sooner in the beginning carcinoma of the larynx is diagnosed and treated by a suitable operation, the more likely the success of the operation, as Frankel and others have repeatedly said.

Cancer of the Larynx—Is It Preceded by a Recognizable Precancerous Condition?

DR. CHEVALIER JACKSON, Philadelphia, read this paper. From clinical observations and the laboratory reports in his cases the following conclusions are reached:

1. While there is here offered some evidence bearing on the histology of precancerous conditions, this evidence is not conclusive.

2. Clinical work is not and never can be ideally perfect. We are human and our patients are human. We cannot expect in our clinical work on the living human being to attain even to the relative certainty of the postmortem room.

3. As laryngologists we are concerned with saving human lives from the inroads of a "dire disease," which is the appellation applied to cancer of the larynx by Sir Henry Butlin.

4. If we admit, as I think we should, that certain curable laryngeal conditions are, in some cases, the sequential predecessors of frequently incurable cancer, it is clearly our duty

not only to eradicate those curable precancerous conditions but to contribute to their early recognition by applying to them the term "precancerous," however faulty such a word may be from a purely scientific, histologic point of view.

5. From a clinical point of view we may regard continual laryngeal irritation from any cause, chronic laryngitis, keratosis, syphilis, pachydermia, socalled prolapse of the ventricle, and benign growths occurring in a person of cancerous age as clinically precancerous, in the sense that they may be contributory factors in the etiology of cancer, and as such should be cured, surgically or otherwise, as may be indicated.

6. It is no argument against this life saving rule to contend that these conditions are too rarely predecessors of cancer to justify regarding them as etiologic factors in cancer. There is no known agent causative of any disease that will always, in all individuals, under average conditions of exposure, produce that disease. The human race would be extinct if such were the case.

7. The time has come for the laryngologist to follow the lead of the general surgeon and the gynecologist in the recognition of the necessity of curing cancer before it starts.

8. There will be fewer deaths from laryngeal cancer when every member of the medical profession fully realizes the frequently malign nature of chronic hoarseness.

Report of a Case of Carcinoma of the Larynx, Laryngofissure.

Dr. Herbert S. Birkett of Montreal, Canada, reported this case of carcinoma in a man aged 58, with a history of hoarseness off and on for one year, being permanent the past four months.

There was an ulceration of the right vocal cord. In outline it was somewhat oval, the inner edge extending downwards. The surface of the ulceration was entirely covered with a whitish, yellow looking slough. The left false cord was swollen, hiding the whole third of the true vocal cord from view. Some immobility of the right vocal cord. Except for some diseased teeth, which were removed, there was no other diseased condition.

One week later a portion of the growth was removed and reported to be a squamous celled carcinoma.

The operation of laryngofissure was done under chloroform anesthesia, morphin and atropin previously injected.

A low tracheotomy was done, the trachea being injected with 10 minims of a 5 per cent solution of cocain before opening. This abolished reflex cough. The division of the allæ of the thyroid cartilage was carried out by a fine saw, the entire vocal cord, with the underlying perichondrium and arytenoid cartilage removed, and the parts sutured with catgut.

Tracheotomy tube removed 24 hours after operation. The patient left the hospital 17 days after operation.

Granulation tissue appeared on the wound. A 50 per cent solution of argyrol was applied and the granulation eventually disappeared.

It is now two and a half years since the operation, and the patient has gained in weight, and his voice has become so much stronger that it can be distinctly heard 160 miles through the telephone.

Section removed showed it to be a squamous cell carcinoma with secondary infection.

Laryngectomy for Cancer With Remarks on Seventy Operated Cases.

By John E. Mackenty, M. D.,
New York.

The operation of total laryngectomy for the cure of intrinsic cancer of the larynx should take a foremost place in the surgery of cancer in general. In no other location in the body have the results excelled, and in a few they have even approached those in the field under discussion.

One of the chief objections to this operation was the loss of voice incident to the removal of the larynx. The fact that the loss of voice was inevitable in the progress of the disease was utterly ignored. Too often the disease was far advanced, owing to the neglect of one single symptom, hoarseness in the middle years of life, an appreciation of which would save thousands af cancer victims.

The factors tending towards an optimistic outlook may be set down as follows:

1. Slowness of growth.
2. Freedom from involvement of the posterior part of the larynx.

3. Superficial growth. Cancer starting in the deeper layers of the larynx may not be more malignant, but they more often escape detection until well developed.

4. Extension forward and downward rather than upward and backward.

5. Age of patient. Cancer in the late thirties or early forties is more malignant than in later life.

Biopsy for diagnosis is to be avoided if possible.

It has been resorted to by the speaker only a few times in eighteen years.

The diagnosis should be made on the history, behavior of the growth, and on the exclusion of syphilis and pulmonary tuberculosis.

Loss of motility in the affected cord is almost a pathognomonic symptom of cancer. He had never seen primary involvement of the interarytenoid.

The extent of the growth must not be estimated by the image seen in the laryngeal mirror, or even by the more accurate impression following direct laryngoscopy. The upper edge of the growth is all that appears for inspection. It would be quite safe to add two-thirds to the visible growth in forming a mental picture of its size.

The speaker is opposed to any opening of the larynx, since the incision may bisect the growth and then disseminate it.

If any doubt exists in the operator's mind, the patient should be given the benefit of that doubt in having the more radical operation done.

Secondary operations for cancer are almost futile in any field. "You have but one shot for a bull's eye, and if you miss the patient pays with his life."

Nor do these operated patients need sympathy, according to the speaker, who has observed nothing but cheerfulness and gratitude on their part at their deliverance from certain death. His experience has led him yearly further away from compromise in dealing with intrinsic cancer of the larynx and has taught him to leave the frankly extrinsic cases to their fate.

The speaker considers the statistics of cures by thyrotomy, and even by excision under suspension, as simply amazing.

Surgery in other fields for cancer strikes wide of the disease. No general surgeon would be content with a quarter of an inch leeway of a malignant growth. His personal position is that thyrotomy is rarely indicated, that radium is on trial and that total laryngectomy is the logical, safe and tried procedure of choice.

The speaker then presented his own statistics showing a percentage of 66 per cent recoveries.

The technic of the operation as performed by the speaker was then presented, the operative techni having been improved in several points, though the rationale of the operation remains the same.

DISCUSSION.

Dr. D. Bryson Delavan, New York: In considering the unsolved problems of laryngeal cancer—the time has certainly arrived, not for theoretical discussion of rival methods, but for such as to indicate a real advance. This is especially true the actual comparison of their practical results.

The value of this symposium, in the speaker's opinion, is of the thesis of Dr. Jackson, which contains much in the way of constructive criticism and suggestion. Prof. Okada's results are particularly encouraging, as, also, is the breadth of view with which he considers the choice of operation, recommending a careful study of the case, and the selection of the operation with reference to its particular character.

Improvements in the methods of diagnosis, which would admit of a clearer vision of the probable course of the growth, would add greatly to the possibilities of successful treatment.

Prof. Okada has emphasized this and has raised the question, Should all cases of laryngeal growth of suspected malignancy be operated upon in the most radical manner, or should discrimination be made, and in certain cases more conservative means be employed?

Experience has taught that in laryngeal surgery no single method has borne off all the honors. Butlin, Semon, St. Clair, Thomson, Jackson, Lynah and others have succeeded well with thyrotomy, while the advocates of laryngectomy have also had their triumphs, as demonstrated today.

One of my patients, operated upon 25 years ago for a partial laryngectomy, is well today. Another, a complete laryngectomy for both intrinsic and extrinsic carcinoma, is without recurrence after six years

Gluck operated upon laryngeal carcinomas of every degree, claiming that he gained an occasional success, and, if the patient died, that he had given him the benefit of an euthanasia.

In 1884, Dr. Solis Cohen established three principles for laryngectomy which have proved of great value.

1. Fixation of the severed end of the trachea externally at the base of the neck.

2. The employment of suitable posture in the immediate care of the case.

3. Most important of all, the value of scrupulous painstaking and untiring watchfulness and care bestowed upon the patient after operation by the operator himself. As a means to diagnosis, biopsy with us is not generally recommended. Dr. Jackson refers to a certain action of the mind as "instinctive diagnosis." The speaker would define this expression as a subconscious reasoning based upon long experience of many closely observed cases. The beginner does not have the instinct for diagnosis. The man of long practice may acquire it. When that instinct or intuition has caused him to believe that a certain condition is what it appears to him to be, it is a wise plan to respect that judgment until disproved.

More than anything else there is needed a collective history and analysis of the results of the treatment of this subject, failures as well as successes, faithfully set forth. Thus it may be learned what methods to imitate, who to avoid and ultimately to prove by what means human life is most likely to be prolonged.

The question of general application that a benign growth may degenerate into one of malignant character has been extensively discussed for many years with negative results.

There is much food for thought in Dr. Jackson's proposition, that there is a curable precancerous state, if we could only recognize it. To this end we would do well to stimulate by all means in our power the efforts of those engaged in the investigation of the etiology of this baffling disease.

Dr. Joseph C. Beck, Chicago, by invitation, said that he was struck favorably and very enthusiastically with Dr. Mackenty's presentation, solely based on his personal experience. He felt that in the future he would do more laryngectomies. The point was brought out that the patient should not be conscious. The last moment of removal of the larynx is most important. That is a dreadful feeling when they hear "now it is out." The question of an anesthetic; the rectal anesthesia of ether and paraldehyde, with immediate and regular and timely administration of morphin, in combination with magnesium sulphate hypodermically; he had not had a single bad result in almost two hundred cases. The patient is relaxed and yet he will cooperate with you, even to the point of attempting to expel secretions of blood that may accidentally run into the trachea. In fact, nothing has given better results than having the patient swallow the duodenal tube.

Regarding Dr. Jackson's paper, he had been thinking of the lively time and the controversy with Dr. Bloodgood and Dr. Leo. Loeb, and he had hoped that Dr. Loeb would be here to speak on the precancerous condition, a term very much attacked by pathologists, and that is what it amounts to.

Dr. Fielding O. Lewis, Philadelphia, said that his experience is limited to six cases of laryngectomy, none performed long enough to determine a cure. As to the extrinsic involvements, it would seem to him that the comfort these cases receive, though they may live only a few months or a year, is sufficient to repay them for what they suffer from the operation. He cited the case of a woman, 58 years of age, with extrinsic involvement, and he removed, together with the larynx, the upper part of the esophagus. The free end of the esophagus was then attached to the skin at the side of the neck. She has been quite comfortable, has gained twenty pounds in weight, does home work for a large family with no signs of recurrence, though it is only six months since the operation.

As to production of voice, he recalled hearing Sir Robert Woods state that he taught his laryngectomy patients to produce sound by administering a few grains of citric acid followed by a few grains of bicarbonate of soda. A carbonic acid gas generated produces a sound as it escapes from the stomach through the esophagus. He has tried this method with fair success. Feeding through a Rehfuss tube, as suggested by Dr. Beck, has been used in his cases, which is introduced into the duodenum.

Dr. Robert C. Lynch, New Orleans, said that he had had fifty-two cases, seventeen cured, fourteen living and the other dead from other causes. The points in the technic of operation resembled Professor Okada's in a way, and in doing the dissection from below upward, and when he cut through the trachea he put in a large rubber tube, such as Dr. Mackenty used, that was sewed into the trachea at the time it is cut through. In that way he was able to protect the trachea from any entrance of blood, and then, after a time, or at the time the wound is dressed, he used these block tin tracheotomy tubes, made unusually large, so they practically fit the trachea closely.

The last six cases done he used this tube instead of a tracheotomy tube. He had used the gas oxygen and ether anesthesia with perfect comfort to the patient. There are two of the cases which have developed speech which could be heard anywhere in this room. One was a doctor, who has returned to his practice of medicine.

As to Dr. Jackson's paper, he wished to speak of one point. He thought we should guard against treating precancerous conditions with radium, and he did not believe we should follow the method of dermatologists in treating with radium. If the thing was treated with radium one was not absolutely sure. He had seen many cases come that had had radium, in which apparently the precancerous condition was stimulated so that it then had an actively growing squamous cell carcinoma. In regard to cases that should have laryngectomy, he grouped all cases much as our Japanese friend has stated, doing thyroidotomy on the early cases and then doing laryngectomy on the extensive cases.

Dr. Gordon B. New, Rochester, Minn., said: Regarding Dr. Jackson's paper, he wished to speak of one point. He thought we should guard against treating precancerous conditions with radium. If such conditions are treated with radium the surface may be scarred over and a cancer extend deeply into the tissues underneath the scar. He had seen many patients who had had radium treatment, and apparently a precancerous condition was stimulated so that at the time the patient came a squamous cell carcinoma was growing actively. These precancerous conditions should be removed surgically.

With regard to cases in which laryngectomy is performed. Thyroidotomy is performed in the early cases and laryngectomy in the more extensive cases. Some patients with low-grade malignancy and gland involvement have been well for many years after block dissection.

DR. E. ROSS FAULKNER, New York, said that Dr. Mackenty has presented a notable record of his conclusions. The evolution of the surgery of the larynx has been, according to Dr. Mackenty, that conservative measures have to be given up for more radical measures. We would all like to evolve in another direction, from radical to more conservative. There is always a stage where conservative measures might do, but the difficulty was to find them. A case came to him where he thought it would be an ideal case for thyroidectomy. He opened the larynx and was quite at a loss to know what to do; three frozen sections were made and they were every one malignant. In this case he did a laryngectomy. It was the only thing to do.

Radium will probably help a great deal in saving cases that before were inoperable, but he did not think it would ever change the type of operation. He thought the radical operation was usually the thing indicated when a diagnosis was made.

DR. CHEVALIER JACKSON, Philadelphia (closing), said that in 1909 he reported fourteen cases of laryngectomy. They are all dead now, and the average duration of life was but little over a year. He felt in regard to that, that if laryngectomy could not show more than a year's duration of life after operation that we had not gained anything. A patient who was in fit condition to stand a major operation like laryngectomy was good for another year without doing anything. Dr. Jonathan Wright and our worthy president reported a case in their classical textbook in which a patient had lived for thirteen years without any operation whatever. The patient may be still living.

In regard to the selection of the procedure that Professor Okada spoke about and that was emphasized by Dr. Delavan, to his mind that was one of the most important things. A cancer limited to the tip of the epiglottis, he thought should be removed, as was accomplished many years ago by Dr. Delavan in the cure of squamous cell epithelioma. When, however, there was a growth on the vocal cord, if we could get the case when limited to the cord, then he felt that the operation was laryngofissure. Distinction should be made between anterior and posterior intrinsic growths. He felt that we should make a third division and subdivide the intrinsic into anterior intrinsic and posterior intrinsic. If close to the posterior portion of the cord, then it was an exceedingly serious and usually fatal disease, a foci disease, as Sir Henry Butlin calls it.

Dr. MacKenty (closing) said that laryngectomy was a safer operation than thyroidectomy. As to the anesthesia, he did not approve of rectal anesthesia. He regretted to hear that Dr. Lynch does a two stage operation, but he was glad to hear him say that radium is bad in these precancerous conditions.

Sarcoma of the Nose.

Dr. Cornelius G. Coakley, New York, presented a patient who had been referred to him a year ago last November by Dr. Mason of this city. She had had two or three intranasal operations for a sarcoma involving the left nasal cavity, supposed to be somewhere in the region of the middle turbinaet. The entire nasal cavity was filled with a mass; it was impossible to tell point of attachment, and he had refused to do anything intranasally and insisted upon an external operation. The operation was done November 18, 1920, and an incision was started from the region of the supraorbital notch and carried down more on the cheek.

From the size of the mass it would probably involve the maxillary sinus as well as the ethmoid region and even possibly the frontal. The whole of the nasal bone was removed. In the nasal fossa of the superior maxilla and ethmoid cells there was a very large mass found, which was grasped with a pair of forceps, and in pulling it out he was surprised to find attached to it a piece of bone that proved to be the whole of the cribriform plate of the ethmoid. The dura was found exposed. There was no involvement of the antrum, no involvement of the frontal sinus. He expected to have a meningitis and a rather quick going out. There was no evidence of headache or meningeal irritation. About three weeks after operation radium was used externally, and up to this present time there was no recurrence within the nose. She was shown because of the lack of facial deforming, that can be gotten through a wide area of exposure through the ethmoid. So that even a fastidious patient cannot object to the cosmetic results of the operation.

The Control of Mandibular Pain Through the Nasal (Sphenopalatine Meckel's) Ganglion.

By Greenfield Sluder, M. D.,
St. Louis.

Since 1908 he had been surprised by phenomena apparently originating in or controllable through the nasal ganglion. Some of them have permitted of explanation more or less on an anatomic basis. Others, however, have not been possible of explanation on such basis, according to our present anatomic knowledge. The control of pain in the lower jaw through or by means of the nasal ganglion belongs to the second category.

The first observation of this phenomenon was accidental. A patient who was subject to recurrent attacks of nasal ganglion neuralgia appealed to him for relief from an attack with which he had become familiar through experience, stating that in addition he had a very painful wound in his lower jaw, where a molar had been dug out with much difficulty. The wound was thirty hours old and inflamed. He anesthetized the nasal ganglion and found that the pain in the wound was stopped as well as the neuralgia.

Since then he had four instances of painful wounds of the lower jaw, made by extraction of teeth, in patients who were not sufferers from nasal ganglion neuralgia. In them the pain was stopped by cocainizing the nasal ganglion. The relief was of several hours' duration and in one it did not return.

The possible therapeutic value of this observation has not been determined.

The Control of Earache Through the Nasal (Sphenopalatine Meckel's) Ganglion.

By Greenfield Sluder, M. D.,
St. Louis.

The relief of earache through or by means of the nasal ganglion apparently falls into the category of those cases of ocular, mandibular and glossal pain that can be relieved in this way. No anatomic facts can be offered as an explanation of these phenomena.

Earache is not an uncommon feature in the syndrome of nasal ganglion neuralgia and may be an isolated manifestation. In these cases examination of the ear is negative in every way. This fact has been mentioned often in the course of the description of that syndrome.

Recently he discovered that the pain of inflammatory disorders of the middle ear in patients who were not sufferers from nasal ganglion neuralgia could be stopped by cocainization of the nasal ganglion. To what extent or with what frequency this may prove true he does not know at present. It appears, however, to be a phenomenon worthy of investigation. Its therapeutic value would seem obvious, were it a fact of reasonable frequency.

He had nine cases of earache accompanied by more or less congestion of membrana tympani, one with obliteration of the landmarks without pouting, two middle ear abscesses with pouting, one general reddening of the membrane, tinnitus, and the others with congestion of the handle of the malleus (eustachian tube cases), that were relieved by cocainization of the nasal ganglion. In one of the abscesses the relief was for a few minutes only. In this case there was reason to think that only a very small part of the cocain was absorbed. In the other case, the relief was complete, but it did not at all prevent the pain of paracentesis. In the case of reddened membrane with tinnitus, the relief of pain and tinnitus was complete with one application and they did not return. This was true of the case with the obliterated landmarks of the membrane and the cases of slight irritation of the eustachian tube.

Dr. Lee Wallace Dean, Iowa City, said: A number of years ago a woman, aged fifty-eight years about, came under his observation, complaining of glossodynia and with the diagnosis of carcinoma of the base of the tongue. Three years before he saw this patient she suffered from mild coryza accompanied by pain, localized on the left side of throat and tongue. In the course of weeks, perhaps months, this pain became localized in the tongue. It was of a neuralgic type.

Examination of the throat was negative. No evidence of any lesion of the tongue. Cocainization of the left sphenopalatine ganglion gave complete relief. Mild sphenoiditis left was found. The left sphenoid was operated upon and the ganglion injected. This gave complete and permanent relief from the pain.

DR. GREENFIELD SLUDER, St. Louis (closing), said: Dr. Dean told him of the observation for the control of glossodynia and since that time he had injected three old and bedfast cases of glossodynia.

To find something that can and does control glossodynia seems to him the most valuable contribution to the subject. Dr. Dean spoke of not being always able to inject the nasal ganglion. Dr. Sluder thought that probably we do not always inject the nasal ganglion. We inject in the vicinity of it and often get results in that way. Experiment on the cadaver showed that a needle put through under the posterior tip of the middle turbinate uniformly landed, if not in, certainly very close to it.

You can always turn around the tip of the middle turbinate and go through the sphenoid foramen. The recommendation that he would make was that the straight needle be abandoned and the curved needle used to curve around the posterior tip of the middle turbinate that can be guided under the pharyngoscope, as advocated by Dr. Edgar M. Holmes some years ago, or after your needle goes through the foramen. You can feel it go through and thus you get anteroposterior play of the needle in soft tissue by something more than an eighth of an inch, probbaly more nearly a quarter of an inch, and he would recommend that procedure for anyone who has trouble with the straight needle.

Carcinoma of the Larynx and Allied States Treated Locally With Radium. A Clinical Report.

BY OTTO T. FREER, M. D.,
CHICAGO.

He described the method of application and a series of 27 cases of carcinoma of the larynx treated by radium emanation and a number of supplementary cases, nasal and laryngeal.

Recurrences.—Among the 27 carcinomas of the larynx treated there were 11 returns of the growths, averaging four and one-half months as the interval between the last intralaryngeal radium emanation treatment and the evidence of the return. Among the recurrent cases were those of six patients who were predisposed to a return because of the advanced state of their cancers when treatment was begun, all of them being of the type called inoperable, with large tumors or very deep infiltration. The eleven recurrences leave of the 27 carcinomas 15 where there was no return. Four recurrences, including one that had made great progress before the patient came back for renewed treatment, have been successfully eliminated by reraying. In another patient, a great tumor that had been permitted to grow a long time by the patient before he returned, and that occupied the base of the tongue, disappeared completely after renewed raying.

Since the beginning of treatments with emanation in the larynx there have been 10 deaths among the cancer patients.

DURATION OF TREATMENTS AND DOSAGE.

Experience shows that a given dose of emanation rayed from a large amount of emanation in a short time is more effective in destroying a growth than the same dose supplied by a smaller quantity during a longer period. To attain a certain intensity of raying requires a corresponding increase in the number of millicuries in the screen used. Within the last three months increase in the radium supply has made trebling the dose of emanation possible so that a treatment that formerly took from two to three hours can be given in one. For intralaryngeal raying this asks less endurance of the patient, cuts down the number of treatments and so the total expense, making them available to those of small means. In the case of a relapse, instead of having to again go through a series of four to five hours of hour-long treatments, two hours of raying will suffice, and for prophylaxis from one-half to one hour will be enough.

REACTIONS.

Reactions consisted of pain, cough, reddening, the formation of a peculiar, white, clinging membrane firmly attached to and infiltrating the epithelial layer of the mucosa and of edema.

While there is much deep cancerous infiltration of the tissues, the edema of reaction may be great and cause dyspnea.

Raying of the larynx through the tissues of the neck from outside, even if intense and prolonged for seventeen hours with from 300 to 500 millicuries of emanation, has too feeble an effect to be reliable.

SELECTIVE ACTION OF THE RADIUM RAYS AND PROGNOSIS.

Unlike the other ways of removing cancer, which, with the exception of the X-ray, destroy the normal with the pathologic tissue, radium possesses the power to take the life of the cancer and other cells of low vitality, while sparing, though lowering temporarily the life of the normal cells. Cancer cells of a very low vitality as those of the rapidly growing soft cancers succumb to astonishingly small doses of emanation that do not perceptibly affect the healthy flesh at all, while unfortunately other cancer cells may possess a vitality almost equal to that of the normal tissues and be but little influenced by great amounts of raying that profoundly affect the healthy tissues and create violent inflammation of long duration. Such resistant cancer cells may be made merely dormant over a variable period by raying and begin to grow again as soon as they have regained their vitality.

Not only the cancer cells vary in their vitality, but the tissue cells also, so that a cancer strongly resistant to it gives a hopeless prognosis. The patient's chances therefore depend largely upon the proportionate vitality of his tissue cells and the cancer cells, and to foresee just what this is cannot be told at present. Where this proportion is favorable to the patient raying gives some marvelous triumphs, and the fading away of the cancer with the emergence of normal parts, especially the white cords with returning motion where fixed, is one of the delightful wonders of practice. It is remarkable how long the cords can endure being imbedded in cancerous masses and edemas and yet remain intact before the strangling pressure of the cancer leads to their complete atrophy and disappearance, an atrophy that takes place usually without ulceration.

PROPHYLACTIC RAYING.

Considering the tendency to recrurence possessed by a proportion of the cases, especially advanced ones, it is advisable to give the patient at intervals of from three to six months a prophylactic treatment of from 200 to 300 millicurie hours.

Selective action of radium and the effectiveness of raying from a fossa pyriformis are strikingly shown in one of the cases.

The experience gained from the cases here described confirms the speaker's opinion that, in spite of possible recurrences, radium emanation, applied intralaryngeally as described, is the best existing remedy for laryngeal carcinoma, either intrinsic or extrinsic. The ready control and removal of cancerous returns and the absence of increased vulnerability of the normal tissues after an interval of three months or more have made the outlook unexpectedly favorable for patients with recurrences if reported early enough.

DR. CHEVALIER JACKSON, Philadelphia, said that he thought Dr. Delavan had some data very much closer related to radium treatment. His experience was very limited, and he would like to hear the data of Dr. Delavan. He quite agreed with Dr. Freer in regard to laryngectomy.

DR. D. BRYSON DELAVAN, New York, said that he thought nothing could have expressed his own views better than those of Dr. Freer's.

DR. CHEVALIER JACKSON, Philadelphia, said that he would like to ask Dr. Delavan a question in regard to technic. He spoke about the preoperative use of radium. What were his views as to the advisability of that, and also whether or not there is to be a definite lapsed period after the raying before the operation, whether it has any effect on the healing? Whether it should be used later before undertaking operation?

DR. CORNELIUS G. COAKLEY, New York, said that at the meeting of the New York State Medical Society in April, this year, there was a symposium on the treatment of cancer of the larynx by three different methods, one on radium by Dr. Quick, another on laryngofissure, and another on laryngectomy. He had asked Dr. Quick if he might have the privilege of seeing some of the results of the radium as to what he would consider favorable cases in the Memorial Hospital. He saw there ten cases selected from the 150 odd which he was able to get to come back. Now, of these ten cases one of them was a sarcoma of the larynx in a woman, and the result there was perfectly wonderful. She had a perfectly clean larynx; the radiation had been done something like two or two and a half years, and she had an excellent voice. She had a perfectly smooth larynx—no evidence of thickening anywhere within the larynx, and certainly that condition, inasmuch as it was a bilateral involvement of the larynx, could not have been accomplished outside of a total laryngectomy, but here was a woman with a good larynx, a good voice and, so far as we could see, no possible evidence of recurrence. We saw also another case, an epithelioma involving the pyriform floor, the epiglottis and the arytenoepiglottic folds. Section had been taken of that case and found to be epithelioma, and was perfectly good, smooth, no nodular, possibly most of the epiglottis was gone; the aryepiglottic fold was in perfectly good shape, and that case had been a period of two or two and a half years since the application of radium. He would regard that as a very excellent result. The third case that he saw was a man with a bilateral involvement of his larynx of somewhat over a year, extending since the last application of radium. He had a very fair voice, husky, the cords were both nodular, slightly reddened, much like some of the cords we see after some very bad papillomatous condition. The condition was excellent.

Dr. Quick showed a case that had been seen in his office, a private patient, some two or two and a half years ago, which he claimed was a cure. The man had an involvement of both sides of his cords and ventricular bands, and he advised him that laryngectomy was the only chance he had of living very long. He positively refused operation. Radium was used and he was shown to me as a cure. He had nothing but a whisper voice; he had in the upper part of his larynx still some thickening in the ventricular bands, cords moderately thickened, and he had a mass extending from the anterior commissure probably into the trachea. He told me that they had made a direct examination of this patient, and what was seen was nothing but a scar tissue. He doubted that, because in such cases where there are practically always carcinomatous cells left in that thickening, and it is practically only a matter of time before recurrence takes place, and so while this man has had two years of life and has been very comfortable, he did not think there is any doubt that time will show that that patient will have a recurrence. Another case was shown. No section taken. Anterior part of right vocal cord was visible and not implicated with any inflammatory or other process, but he had in the region of the arytenoid a swelling as large as in the sixth chart here, as big as that, edema. It does not seem to me that sufficient has been accomplished yet.

Dr. Coakley said that was the one in which no section was taken, and you, Dr. Freer, said the cord was perfectly normal. Now, within a year there was a lesion on the cord at the same site, and he could not help feeling that lesion was a return of the same process that was going on, and whether a larger dose was going to destroy the remaining cells, it was going to destroy all the cells, or was it going to leave some that will lie dormant a year or two and then start up again?

Now, before radium came into use he could not recall of a single case of carcinoma of the larynx, hypolarynx, in which the patient died of metastasis in other portions of the body. Perhaps his clinical experience was different from others. But with the use of radium, especially as used years ago, in smaller doses, before we knew as much about it as we do now, he had seen a number of cases of metastasis. It was unfortunate to report cures in a few months from the action of this great agent.

Dr. Henry L. Swain, New Haven, said that he supposed that Dr. Coakley would not want his whole larynx taken out if he had a small growth on the cord. Every patient would prefer to have the other possibilities in the way of cure properly exploited first. If the question he was about to propound could be answered definitely, he would ask Dr. Freer and Dr. Delavan what evidence there was that the healing of the tissues after the exposure to X-ray has been at all disturbed? He meant that if one had radium used to the extent of attempting to destroy a growth and then wanted to have an operation, has there been any damage done to the tissues by its use? He did not quite agree with all that Dr. Coakley said, although we must in the face of things consider very earnestly what he has propounded. We have ocular demonstration of metastasis subsequent to absorption by radiation. It is a matter of extreme importance to decide if the tendency to metastasis in all malignant growths is at all increased by destruction through radiation. We now know most certainly that these distant tumors are not prevented by this agent.

Dr. Lewis, Philadelphia, said: At the radiologic clinic of the Philadelphia General Hospital, which has only been in operation since February, we have been burying emanations (as he understands this is the method used at the Memorial Hospital in New York) into the growth, whether it be in the larynx or the base of the tongue. It would seem to him that this method is perhaps as efficacious and less uncomfortable to the patient than using an applicator such as Dr. Freer has described. He was much pleased to hear him state his results in the treatment of nonmalignant growths of the larynx. He has had two very remarkable results, in which radium needles were inserted into the larynx. Both have been operated on repeatedly for the removal of papilloma. After application of radium it would be almost impossible to state that the patient had ever had any laryngeal growth.

As to the technic, he would like to ask Dr. Freer if the emanations or the salt of radium was used in conjunction with an applicator?

Dr. D. Bryson Delavan, New York, replied to Dr. Jackson's question as to when radium should be applied in operative cases, and stated that it is the custom in Memorial Hospital, New York, to apply radium before operation. He agreed with Dr. Freer that the study of radium is still in the experimental stage.

The use of the word "cure" is unfortunate. Some of the older operators claimed a cure if there was no recurrence in one and one-half years after operation. The speaker mentioned a case of recurrence nine years after operation, and cited the case reported yesterday in which recurrence of carcinoma had taken place twenty years after a successful laryngectomy. How can it be known that any case is "cured" and secure from recurrence, whether treated surgically or by any other means? Only time can prove this.

Dr. Douglas Quick, the speaker's associate at the Memorial Hospital in New York, has prepared a review of 156 cases treated with radium at the above hospital during the last five years. His conclusions, based upon elaborately studied material, have been worked out with all possible accuracy and care. His impressions of conditions observed have been confirmed by other competent examiners. In reporting successful cases the author has avoided the use of the term "cured."

The speaker then presented the following summary of conclusions of this study by Dr. Quick:

1. While radium offers a hope to a larger number of cases of cancer of the larynx than the other methods, its use must be considered as yet, to a certain extent, experimental.
2. Before treatment of a laryngeal neoplasm is undertaken proper classification, based upon what can reasonably be hoped for should be made and the method and intensity of treatment governed accordingly.
3. While treatment of primary operable intrinsic cancer of the larynx is permissible, the evidence to date does not warrant advocating it as the agent of choice.
4. It is suggested that the preoperative use of radium in operable cases would add materially to the end result.
5. Surgical exposure may frequently be used to advantage in radium localization.
6. The radical use of intensive radiation is permissible in cases offering a reasonable hope of complete recovery.
7. The conservative use of radiation in inoperable cases offers palliative relief in a large percentage.
8. Radium should be withheld in the very advanced cases.

Dr. Freer asked Dr. Delavan what the dosage of the emanations used was.

Dr. Delavan: The dosage depends altogether upon the case.

Dr. Freer: And how applied and where?

Dr. Delavan: Extrinsic, intrinsically mainly through emanations. Many were treated with large doses extrinsically.

Dr. Freer said that his claim, of course, was that this method, which he presented had the merit of exactness in two directions: First, the exact application of the screen to the diseased part, which was highly important, so that the diseased part gets more inetnse radiations, or, secondly, ability to change the dose. There is no comparing little doses with tremendous dosage of radium in the refractory cases He was sure that in the larynx 100 mc. will produce a moderate reaction. He knew 300 would produce tremendous reaction.

Dr. Cornelius G. Coakley, New York, said that these implantations done in small capsules are done under direct examination, where they can be accurately placed in the mass at whatever situation, and they are introducing several—anywhere from two, four to five, in accordance with the size of the mass. There can be no question as to where he is going to introduce them; he does it accurately.

Dr. Freer: Does he use suspension?

Dr. Coakley: No, direct tubal examination.

Dr. Freer: He doesn't use a mirror?

Dr. Coakley: He does use a mirror to some extent, but trusts considerably to direct examination of the larynx. They use a broad spatula; he lifts the epiglottis, which gives you an excellent view of it, and sometimes under local and sometimes under general anesthesia. Is that right, Dr. Delavan?

He is under the impression that much larger doses put into some sort of container, shaped much as your container but not held in that position by a mechanical instrument but held there at the time of application by an assistant, guided by a mirror and much of it by direct method but with large doses that he has, he believed five to twenty to thirty minutes was sufficient.

Dr. Freer was delighted to hear Dr. Coakley was reaching the same conclusions that large doses of emanation are the correct thing.

Experiments With Atmospheric Pressure in the Maxillary Sinus.

Dr. Rockwell A. Coffin, of Boston, read this paper.

The question of demonstrating the atmospheric pressure in the sinus maxillare was first suggested to the writer and his assistants by two cases presenting subjective symptoms of antral infection in which the transillumination, Roentgen findings and experimental puncture were negative.

While it is not within the scope of this paper to consider the cause of the various phenomena found during these experiments, we are tempted to enumerate such causes as suggest themselves. Where the maxillary sinus is fairly large and the ostium is extremely small or occluded, the amount of residual air is considerable. This air gradually becomes warmer from the heat of the body, which would make it many degrees warmer than air of a temperature of say, 70 degrees, passing through the nose. Likewise this retained air would take up a percentage of moisture, which, if the ostium were extremely small or obstructed, would produce a slight pressure. Gases about the roots of defective teeth, sometimes discharging into the sinus, tend to increase the slight pressure already present, especially if there is coexisting obstruction of the ostium with no accessory openings. Several cases have shown that the pocket formed at times back of the anterior tip of the middle turbinate seems to be an additional cause of increased pressure in the sinus. Since we did not find negative pressure in any of the maxillary sinuses when respiration was at rest, because of the close proximity of the openings, it seemed probable that much the same conditions existed in the frontal and anterior ethmoid cells, provided they were not connected with the posterior ethmoid cells. However, further investigation will be necessary to prove this. If negative pressure exists for any length of time in the maxillary sinus it must be a rather rare occurrence, unless air is passing through the nose. That these findings have some practical bearing on a few obscure cases in which there is a pain about the antrum would seem to be shown in the following two cases.

These two cases would seem to show, beyond any reasonable doubt, that high atmospheric pressure in the maxillary sinus was the cause of the pain, for in the first case the pressure was removed by opening the antrum under the inferior turbinate, and in the second case the pressure was reduced by the escape of air through the ostium.

In no case, whether normal or abnormal, were we able to demonstrate any constant partial vacuum when respiration was at rest.

Positive pressure was the rule during exhalation, except when the ostium was blocked.

Negative pressure was the rule during inhalation, except when the ostium was blocked.

It is possible for marked positive pressure to exist without secretion or pus, when the natural opening is obstructed.

Symptoms of pain may result from positive pressure, without the presence of liquid in the sinus, if there is any obstruction of the ostium from any cause whatsoever.

Cancer of the Larynx.

DR. PERRY G. GOLDSMITH, Toronto, Canada, said that twenty-five years ago Dr. Henry Butlin removed the right vocal cord for carcinoma. The man remained well until a year ago, when he felt a hard and fixed mass below the angle of the jaw. I saw the man. His tonsil projecetd forward; the mass was apparently external and posterior, and no ulceration was to be seen, and he had no complaint regarding the tonsil. Eventually he died and this is his larynx. A section for examination was made through the tongue; the anterior pillars were involved, and the lingual area has been sectioned also. Both areas were definitely carcinomatous. This shows the larynx twenty-five years after he has had thyrotomy, and I think that in itself is very unique. It also shows the larynx of a man after he has been cured of carcinoma in another region.

INDEX OF AUTHORS.

ADAMS, C. J., 984.
Andrews, A. H., 947, 1139.

BALLENGER, H. C., 899.
Barnes, Geo. E., 107, 1137.
Baum, H. L., 371.
Beck, J. C., 579, 580, 581, 1139.
Boot, G. W., 559, 899.
Bulson, A. E., 131.
Bunch, C. C., 617.

CAVANAUGH, J. A., 899.
Cayce, E. B., 595.
Chamberlin, W. B., 423.
Coakley, C. G., 1171, 1209.
Cobb, E., 523.
Coffin, R. A., 1220.
Cullom, M. M., 1163.

DEAN, L. W., 617.
Dickie, J. K. M., 683.
Donovan, J. A., 176.
Downey, J. W., 1009.
Dworetzky, J., 311.

EZELL, H., 593.

FENTON, R. A., 214.
Fowler, E. P., 488.
Freer, O. T., 1212.

GITTINS, T. R., 507.
Gleason, E. B., 552
Goldsmith, P. G., 1222.
Goldstein, M. A., 264.
Goodale, J. L., 882, 1183.
Grove, W. E., 913, 1143.

HARKNESS, G. F., 964.
Harris, T. J., 192.
Hasty, F. E., 596, 598.
Hastings, H., 157.
Hertzler, A. E., 1013, 1032.
Holinger, J., 588.
Hubbard, T., 1167.
Hurd, L. M., 167.
Husik, D. N., 345, 1039.

IGLAUER, S., 105.
Ingersoll, J. M., 1123.
Israel, J., 543.

JACKSON, C., 1182, 1200.

KENYON, E. L., 244.
Keeler, J. C., 759.

LEMERE, H. B., 204.
Levy, L., 493.
Lewis, F. O., 1171.
Lewy, A., 239, 558, 1154.
Lillie, H. J., 990.
Loeb, H. W., 273.
Long, C. H., 573.

McDERMOTT, V. D., 266.
McKenty, J. E., 1101, 1202.
McKinney, R., 977.
Minton, J. P., 563.

NORCROSS, E. P., 501, 574.
Novak, F. J., 878.

OKADA, W., 601, 1197.

PIERCE, N. H., 899.
Pilot, I., 231, 252.
Pohlman, A. G., 1, 430.
Pollock, H. L., 575, 576, 577, 579, 581.
Pratt, J. A., 46.
Proetz, A. W., 1083.

RAE, J. B., 546.
Richardson, C. W., 678, 960, 1166, 1193.
Rackemann, F. M., 1185.
Rosenheim, S., 1027.
Rundstrom, J. A., 950, 1148.

SABINE, P. E., 561.
Salinger, S., 220, 254.
Sewall, E. C., 352.
Seydell, E. M., 862.
Shambaugh, G. E., 225, 266.
Shea, J. J., 813.
Shemley, W. G., 869.
Skillern, R. H., 813, 997, 1175.
Sluder, G., 172, 1209.
Smith, R., 1183.
Sonnenschein, R., 137.
Stark, E. K., 329.
Stein, O. J., 242, 1128, 1156.
Strauss, J. F., 715.
Stucky, J. A., 200.
Sutton, D. C., 1155.

TEFFT, L. E., 329.
Theisen, C. F., 1118.
Trinder, J. H., 1044.

VAN DER HOEVE, J., 279.
von Colditz, G. T., 1161.
Voorhees, I. W., 895.

WALKER, I. C., 660, 1184.
Weller, C. V., 110.
Wilson, J. G., 563.
Wood, G. B., 1191.

ZEBROWSKI, A., 335, 739.

INDEX OF TITLES.

INDEX OF SUBJECTS

ABSCESS brain, Adams, 984; Dickie, 683.
Abscess extradural, Skillern, 997, 1175.
Abscess larynx, Theisen, 1118.
Abscess lung, Richardson, 960.
Abscess retropharynx, Smith, 1183.
Abscess trachea, Theisen, 1118.
Adenocarcinoma antrum, Husik, 345.
Adenocarcinoma ethmoid, Husik, 345.
Adenocarcinoma middle turbinate, Husik, 345.
Adenoiditis, Salinger, 220, 254.
Adenoids, Pilot, 231, 252.
Alcohol injection, Stein, 1128, 1150.
Anesthetic local, Bulson, 131; Hurd, 167.
Angina Vincent's, Husik, 1039.
Antrum adenocarcinoma, Husik, 345.
Asthma, Rackemann, 1185.
Atrophic rhinitis, Pollock, 577; Rundstrom, 950.
Audition, Pohlman, 1, 430; Wilson and Minton, 563.
Aviation, Tefft and Stark, 329.

BARANY, Voorhees, 895.
Brain abscess, Adams, 984; Dickie, 683.
Bronchoscopy, Hubbard, 1167.
Bronchus foreign body, Pollock, 575.

CANCER larynx, Beck, 580; Freer, 1212; Goldsmith, 1222; Hertzler, 1032; Jackson, 1200; McKenty, 1101, 1202; Novak, 878, 908; Okada, 601, 1197.
Cancer temporal bone, Zebrowski, 739.
Cholesteatoma, Hasty, 596.

DACRYOCYSTITIS, Richardson, 1166.
Deafness, Goldstein, 264; McDermott, 266; Shambaugh, 225.
Dichloramin, Fenton, 214.
Drainage, gravity, Barnes, 107.
Drum ear retraction, Fowler, 483.
Dystrophy pituitary, Beck, 579.

EARACHE, Sluder, 1209.
Ear drum retraction, Fowler, 483.
Ear irrigation, Barnes, 107.
Ear larvae, Zebrowski, 335.
Ear mechanics, Pohlman, 1, 430.
Ear middle, skin grafts, Israel, 543.
Ear plugs, Tefft and Stark, 329.
Ear surgery, Gleason, 552.
Emphysema, Rosenheim, 1027.
Eosin-silver solution, Lewy, 1154.
Epithelioma tongue, Lewy, 558.
Esophagoscopy, Hubbard, 1167.
Esophagus stenosis, McKinney, 977.
Ethmoid adenocarcinoma, Husik, 345.
Ethmoid cells, Andrews, 947, 1139; Harkness, 964.
Extractor, shawl pin, Jackson, 1182.
Extradural abscess, Skillern, 997, 1175.
Eye complications, Harkness, 964.

FATALITIES, Loeb, 273.
Forceps foreign body, Boot, 899.
Fracture skull, Ezell, 593.
Frontal sinus, Lewis, 1171.
Frontal sinusitis, Levy, 493; Skillern, 997, 1175.

GROWTHS nose, Baum, 371.
Growths sinuses paranasal, Baum, 371.

HAY fever, Walker, 660, 1184.
Hearing aids, Sabine, 561.
Hearing device, Shambaugh, 225, 266.
Hearing tests, Dean and Bunch, 617.
Histopathology nose, Baum, 371.
Hyoscin, Hurd, 167.
Hyperesthetic rhinitis, Beck, 581.

INCISOR retropharyngeal abscess, Smith, 1183.
Industrial disease, Donovan, 176.
Instrument new, Hasty, 598.
Intracranial symptoms, Levy, 493.
Irrigation ear, Barnes, 107.

KNIFE drum membrane, Barnes, 1137.

LABYRINTH suppuration, Cobb, 523.
Labyrinthitis, Boot, 559.
Larvae ear, Zebrowski, 335.
Laryngectomy, Beck, 580; McKenty, 1101, 1202.
Laryngitis, Cayce, 595.
Laryngopharynx lipoma, Beck, 1139.
Laryngopulmonary tuberculosis, Dworetzky, 311.
Laryngostomy, Sewall, 352.
Larynx abscess, Theisen, 1118.
Larynx cancer, Beck, 580; Freer, 1212; Goldsmith, 1222; Hertzler, 1032; Jackson, 1200; McKenty, 1101, 1202; Novak, 878, 908; Okada, 601, 1197.
Larynx stenosis, Sewall, 352.
Larynx tuberculosis, Cullom, 1163.
Lateral sinus thrombosis, Pollock, 576.
Lipoma laryngopharnyx, Beck, 1139.
Lung abscess, Richardson, 960.

MANDIBLE pain, Sluder, 1209.
Mastoid cells, Proetz, 1083.
Mastoid operation, Fenton, 214; Hasty, 596; Rae, 546; Stucky, 200.
Mastoid process, Downey, 1009; Keeler, 759.
Mastoid skin grafts, Israel, 543.
Mastoiditis, Norcross, 501, 574.
Maxillary sinus, Coffin, 1220; Grove, 913, 1143.
Maxillitis, Lemere, 204.
Middle ear skin grafts, Israel, 543.
Middle ear suppuration, Lillie, 990.
Morphin, Hurd, 167.
Mosher operation, Richardson, 1166.

NASAL reconstruction, Pratt, 46.
Nasociliary neuralgia, Sluder, 172.
Neuralgia nasociliary, Sluder, 172.
Neurosis nasal, Stein, 1128, 1156.
Nose anatomy, Ingersoll, 1123.
Nose growths, Baum, 371.
Nose infection, Gittins, 507.
Nose obstruction, Gittins, 507.
Nose sarcoma, Coakley, 1209.
Nose suction, Iglauer, 105.
Nose tuberculosis, Chamberlin, 423.
Nose vestibule infection, Hurd, 489.

OCCUPATIONAL disease, Donovan, 176.
Optic nerve, Van der Hoeve, 297.
Otitic pyemia, Seydell, 862.
Otitis media, Barnes, 107; Lillie, 990; von Colditz, 1161.
Otitis media suppurative, Hasty, 596.
Otolaryngology; Holinger, 588; Lewy, 239.
Ozena, Rundstrom, 950, 1148.

PARAFFINOMA, Pollock, 581.
Paranasal sinus growths, Baum, 371.
Petrosal sinus thrombosis, Shemley, 869.
Pituitary dystrophy, Beck, 579.
Postgraduate facilities, Stein, 242.
Pulsating artery pharynx, Pollock, 579.
Pyemia otitic, Seydell, 862.

RADIUM, Freer, 1212.
Radiotherapy, tonsil, Trinder, 1044.
Reconstruction intranasal, Pratt, 46.
Respiration, Wright, 1183.
Retropharynx abscess, Smith, 1183.
Rheumatism, Hastings, 157.
Rhinitis atrophic, Pollock, 577; Rundstrom, 950, 1148.
Rhinitis hyperesthetic, Beck, 581; Stein, 1128, 1156; Sutton, 1155.

SARCOMA nose, Coakley, 1209.
Schwabach test, Sonnenschein, 137.
Sebaceous glands infection, Hurd, 489.
Septum deflection, Pratt, 46.
Sinus frontal, Lewis, 1171.
Sinus lateral thrombosis, Pollock, 576.
Sinus maxillary, Coffin, 1220; Grove, 913, 1143.
Sinus paranasal growths, Baum, 371.
Sinus petrosal thrombosis, Shemley, 869.
Sinus Sphenoid, Harkness, 964.
Sinus thrombosis, Seydell, 862.
Sinuses paranasal, Coakley, 1171; Ingersoll, 1123; Proetz, 1083; Skillern, 855; Van der Hoeve, 297.
Sinusitis frontal, Levy, 493; Skillern, 997, 1175.
Sinusitis sphenoethmoiditis, Shea, 813.

Skin grafts mastoid, Israel, 543.
Skin grafts middle ear, Israel, 543.
Skull fracture, Ezell, 593.
Spatula drum membrane, Barnes, 1137.
Speech defects, Kenyon, 244.
Sphenoethmoid sinusitis, Shea, 813.
Sphenoid instrument, Cavanaugh, 899.
Sphenoid, pulsating, Long, 573.
Sphenoid sinus, Harkness, 964.
Sphenopalatine ganglion, Sluder, 1210.
Stenosis esophagus, McKinney, 977.
Stenosis larynx, Sewall, 352.
Stenosis trachea, Sewall, 352.
Suction, Iglauer, 125.
Suppurative otitis media, Hasty, 596.

TEETH in septum, Ballenger, 899.
Temporal bone, Keeler, 759.
Temporal bone cancer, Zebrowski, 739.
Temporal bone skiagraph, Pierce, 899.
Thrombosis lateral sinus, Pollock, 576; Seydell, 862.
Thrombosis petrosal sinus, Shemley, 869.
Thyrotomy, Hertzler, 1013.
Tic douloureux, Lemere, 204.
Tinnitus, Harris, 192.
Tongue epithelioma, Lewy, 558.
Tonsil, Gittins, 507.
Tonsil hypertrophy, Trinder, 1044.
Tonsil radiotherapy, Trinder, 1044.
Tonsil tubercle, Weller, 110.
Tonsillectomy, Hastings, 157; Richardson, 678, 1193; Rosenheim, 1027; Wood, 1191.
Tooth in bronchus, Pollock, 575.
Trachea abscess, Theisen, 1118.
Trachea lymphatics, Strauss, 715.
Trachea stenosis, Sewall, 352.
Tubercle tonsil, Weller, 110.
Tuberculosis laryngopulmonary, Dworetzky, 311.
Tuberculosis larynx, Cullom, 1163.
Tuberculosis nose, Chamberlin, 423.
Turbinate middle adenocarcinoma, Husik, 345.

VASOMOTOR disturbance, Goodale, 882, 1183.
Vestibule nose infection, Hurd, 489.
Vincent's angina, Husik, 1039.
Vocational disease, Donovan, 176.
Voice, Wright, 1183.

19
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